In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 10-410V
(To be Published)

* *		
	*	Special Master Corcoran
SHEELA BLACKBURN,	*	
	*	Filed: January 9, 2015
Petitioner,	*	
	*	
v.	*	
	*	Entitlement Ruling; Human Papillomavirus
SECRETARY OF HEALTH	*	("HPV") Vaccine; Guillain-Barré
AND HUMAN SERVICES,	*	Syndrome ("GBS"); Chronic Inflammatory
	*	Demyelinating Polyneuropathy ("CIDP");
Respondent.	*	Molecular Mimicry; Homology; Onset
	*	
* *		

Isaiah R. Kalinowski, Maglio, Christopher & Toale, Washington, DC, for Petitioner.

Ann D. Martin, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On June 30, 2010, Sheela Blackburn[2] filed a petition seeking compensation under the National Vaccine Injury Compensation Program[3] (the "Vaccine Program") alleging that she suffered a variant of Guillain-Barré syndrome ("GBS") caused by her receipt of the Human

[1] Because this decision contains a reasoned explanation for my action in this case, it will be posted on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, § 205, 116 Stat. 2899, 2913 (codified as amended at 44 U.S.C. § 3501 note (2006)). As provided by 42 U.S.C § 300aa-12(d)(4)(B), however, the parties may object to the decision's inclusion of certain kinds of confidential information. To do so, Vaccine Rule 18(b) permit each party 14 days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the decision will be available to the public. *Id.*

[2] The case was originally titled *Sheela Roten v. Sec'y of Health & Human Servs*., but the caption was changed by Order dated March 1, 2013 (ECF No. 50) after Petitioner changed her legal name.

[3] The National Vaccine Injury Compensation Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (codified as amended, 42 U.S.C.A. § 300aa-10 – 34 (2006)) [hereinafter "Vaccine Act" or "the Act"]. Individual sections references hereafter will be to § 300aa of the Act.

Papillomavirus ("HPV") vaccine on July 23, 2009. Petition (ECF No. 1) at 1. An entitlement hearing in this matter was conducted in Washington, DC on March 25 - 26, 2014.

The case presents two intertwined questions: (a) whether Ms. Blackburn suffered from GBS, and (b) whether Ms. Blackburn's illness began before she received the HPV vaccine. After hearing the testifying witnesses and reviewing the parties' various pre- and post-trial submissions, and based on a review of the entire record as required by the Vaccine Act (§ 300aa-13(a)(1)), I hereby rule in favor of Respondent, for the reasons set forth below.

I. Factual Background and Medical History

A. *Petitioner's Pre-Vaccination Medical History*

Before Ms. Blackburn ever received the vaccination that is the basis for her claim, she had visited healthcare professionals complaining of symptoms similar to those she alleges resulted from the vaccination, although their etiology was at that time unclear.

On October 24, 2008, Ms. Blackburn visited a chiropractor, Mark Snow, and complained of left hip and shoulder pain. Pet'r's Ex. 4 at 2-3. In the course of that examination, she reported constant numbness in her feet and frequent numbness in her hands. *Id.* Thereafter, on November 6, 2008, Ms. Blackburn was seen at the Exodus Health Care Magna Clinic in Magna, Utah, where she reported numbness in her feet over the prior four months, plus more recent numbness and tingling in her hands. Pet'r's Ex. 2 at 15-16. Ms. Blackburn also indicated that she was experiencing nerve pain both in her left shoulder and hip area, which she claimed to be an ongoing problem. *Id.* at 15. She received a diagnosis of a "pinched nerve in lower back, neck, and hip arthritis," but unspecified polyarthritis and neuralgia/neuritis were listed as other possible sources of her symptoms. *Id.* at 15-16.

A few days later, on November 13, 2008, Ms. Blackburn went back to the Exodus Clinic and was seen by Dr. Jill McBride. Pet'r's Ex. 2 at 14. The assessment recorded at the time of this visit indicated that Ms. Blackburn was believed to be suffering from an unspecified polyarthritis as well as neuralgia/neuritis. *Id*.

Based on Dr. McBride's referral, Ms. Blackburn saw Dr. Susan Zimmerman in December of 2008 at the Granger Medical Clinic in Salt Lake City for a neurologic consultation. Pet'r's Ex. 8 at 18. At that time, Petitioner again represented she had been experiencing shoulder pain and paresthesias for the past four months. *Id.* Ms. Blackburn's neurology examination was significant for revealing "mild sensory loss and left-side hyperalgesia," although she presented normal reflex responses. *Id.* at 19. Because the precise cause of Ms. Blackburn's symptoms remained unclear, Dr. Zimmerman made a number of treatment and diagnostic recommendations. In particular, Dr. Zimmerman ordered magnetic resonance imaging ("MRI") of Petitioner's brain

and cervical spine "[t]o help exclude problems such as demyelination, cervical cord compression, tumor or other focal lesions," and raised the possibility of "proceed[ing] to a lumbar puncture or nerve conduction studies to look for other central or peripheral causes of numbness as well." *Id.* The MRIs ordered by Dr. Zimmerman were performed on December 31, 2008. *Id.* at 10-12. The brain scan results were essentially normal, and the MRI of Petitioner's cervical spine revealed "minimal to mild degenerative changes." *Id*.

Less than a month later, on January 14, 2009, Ms. Blackburn had a follow-up visit with Dr. Zimmerman. Pet'r's Ex. 8 at 7-8. Petitioner indicated that she was still experiencing significant right shoulder pain as well as some numbness and pain down both legs (which she attributed to sciatica) plus foot numbness. *Id.* at 7. Upon physical examination, Dr. Zimmerman found that Petitioner had "very mild distal sensory loss." *Id.* He recommended physical therapy to relieve some of the pain that Petitioner was experiencing. *Id*. Additionally, Dr. Zimmerman informed Petitioner about the potential of undergoing nerve conduction studies in an attempt to identify the source of her pain, but Petitioner decided to wait and see if her symptoms improved with physical therapy. *Id*. Accordingly, such testing was never performed until well after Ms. Blackburn's vaccination.

B. *July 2009 HPV Vaccination and Subsequent Medical History*

Petitioner received an injection of the Gardasil vaccination in her left deltoid on July 23, 2009, during a comprehensive medical examination at Exodus Health Care Clinic performed by Cathy Baxter, APRN. Pet'r's Ex. 6 at 1. The past medical history section of the records from that visit, reported that Ms. Blackburn previously experienced an unspecified polyarthritis as well as neuralgia/neuritis. Pet'r's Ex. 2 at 11. Ms. Blackburn also reported "in [the] past couple of weeks having tingling in legs and feet," although her reflexes were tested and recorded to be within normal limits, as well as her cranial muscle responses. *Id.* at 12.

On August 7, 2009, Ms. Blackburn again presented to Exodus where she was seen by Nurse Baxter. Pet'r's Ex. 2 at 9. Petitioner indicated that she had been fine until just recently but was now experiencing "*worsening* tingling in legs and weakness," with her legs feeling heavy as well. *Id*. (emphasis added). As the contemporaneous medical records note, there had been "no findings on all workup last year" as to the source of her condition, although the records again referenced unspecified polyarthritis as well as neuralgia/neuritis. *Id.* Ms. Blackburn obtained a note for light duty at work due to her weakness and discomfort. *Id.* at 10.

A few days later, on August 11, 2009, Petitioner returned to Dr. Zimmerman (the neurologist she had seen at the beginning of the year) for a "semi-urgent follow-up" regarding her ongoing numbness and pain. Pet'r's Ex. 8 at 4-5. Petitioner reported that "[s]he was doing reasonably well until [three] weeks ago," but that she was now experiencing "complete numbness from her knees down bilaterally" as well as back pain and muscle spasms and some less severe right shoulder pain. *Id*. at 4. Notes from the neurological exam performed by Dr.

Zimmerman indicate that the exam was "notable for areflexia[4] and ankle dorsiflexion weakness bilaterally." *Id*. Dr. Zimmerman also stated that "no cause of her pain has yet clearly been identified," but that the onset of Ms. Blackburn's symptoms occurred in the "context of increased stress" given that Petitioner worked two jobs. *Id.*[5] No connection between Ms. Blackburn's July 23rd vaccination and her symptoms was made at this time.

In order to diagnose the cause of Ms. Blackburn's symptoms, Dr. Zimmerman recommended a MRI of the lumbar spine plus a lumbar puncture (in light of her demonstrated areflexia) to "look for the cyto-albuminologic dissociation that can be seen in Guillain-Barré syndrome," but Petitioner refused to undergo the latter. Pet'r's Ex. 8 at 4-5. The MRI did not reveal the presence of any abnormalities. *Id.* at 2-3.

Ms. Blackburn thereafter went back to her chiropractor, Mark Snow, on August 17, 2009, complaining of continued pain in her left shoulder as well as the previously-reported numbness in her hands and feet. Pet'r's Ex. 4 at 4. Deep tendon reflex ("DTR") testing performed during the visit revealed normal reflexes in her upper body limbs, but areflexia in her knees and right ankle. *Id.* at 5. Cranial nerve testing, however, revealed no abnormalities, and the record from this visit specifically indicated that Ms. Blackburn was "able to perform the normal range of facial movements, no asymmetry or other abnormalities were noted." *Id.*

Ms. Blackburn saw Mark Snow again two days later, at which time she underwent a nerve conduction velocity study, the results of which were abnormal. Pet'r's Ex. 4 at 7. Results from that study and an electromyography ("EMG")[6] were reviewed by Robert A. Sellin, PT, DSc, ECS, a board certified electromyographer. *Id.* at 12. Those nerves tested had normal amplitudes, latencies, and nerve conduction velocities, but there was no measurable sensory response from any of the sensory nerves tested; the left median motor distal latency was severely prolonged and the distal nerve conduction velocity was markedly slowed; the left ulnar motor distal latency was severely prolonged and the segmental nerve conduction velocities were markedly slowed; bilateral fibular (peroneal) motor responses were not obtainable; bilateral tibial motor evoked responses were of markedly diminished amplitude and exhibited severe temporal dispersion; bilateral common fibular (peroneal) and tibial motor F-waves were not obtainable; and the left median and ulnar motor F-waves were severely prolonged. *Id.* The remarks included

[4] Areflexia is defined as the absence of reflexes. *Dorland's Illustrated Medical Dictionary* (32d ed. 2012) at 130 [hereinafter "Dorland's"].

[5] The medical records from this visit with Dr. Zimmerman also reflect that Petitioner reported that "[five] weeks ago [*i.e.* some time in July 2009] she had symptoms similar to a friend, who was diagnosed with strep throat,' although "[s]he did not seek medical attention at that time." Pet'r's Ex. 8 at 4.

[6] Electromyography ("EMG") is "an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation." *Dorland's* at 602.

with the testing results indicated that "[t]hese electrophysiologic findings combined with her history are strongly suggestive of some form of acute inflammatory demyelinating polyradiuloneuropathic process (AIDP)[7] such as Guillain-Barre syndrome)." *Id.* at 8. The remarks also indicated that "I believe it is important to note that the patients symptoms were very mild [six] weeks ago and she received immunization about [five] weeks ago and then noticed her *increasing* symptoms. *Id.* at 9 (emphasis added). The EMG results revealed that "[a]ll muscles tested exhibited normal insertional activity, electrical silence at rest, and had normal motor unit configuration and recruitment, except: There was reduced interference patterns in all muscles tested except the left deltoid." *Id*. at 9-10.

On August 24, 2009, Ms. Blackburn went to the University Health Care Emergency Department at the University Health Care Hospitals and Clinics in Salt Lake City, Utah complaining of three and a half weeks of progressive ascending weakness and numbness as well as back pain, and noting that her condition had occurred after receipt of the Gardasil Vaccine. Pet'r's Ex. 3 at 19. She specifically reported that she last felt well on July 4th when she became ill with flu-like symptoms, and then later that month received the Gardasil vaccination. *Id.* During this visit, Ms. Blackburn noted that for the first two weeks her symptoms were mild and that she had seen Dr. Zimmerman, but her symptoms had progressed to the point where she was having difficulty walking. *Id*. The contemporaneous medical records from this visit included GBS and multiple sclerosis ("MS") as potential differential diagnoses. *Id.* at 42.

The next day Ms. Blackburn saw Dr. Jeffrey C. Wagner, a neurologist. Pet'r's Ex. 12 at 35-36. She informed Dr. Wagner that she had been using a walker since her visit to Dr. Zimmerman earlier in August, and that she was experiencing more tingling, numbness, and weakness in her hands and arms. *Id.* at 35. Petitioner again reported having experienced "strep or flu-like symptoms at the beginning of July" with "associated neck stiffness," but she noted that these symptoms had improved and "she was feeling back to her usual state of health by the end of July when she presented for a general annual physical exam" and "received the Gardasil vaccination." *Id.* at 35-36. After examination of Petitioner and review of her history, Dr. Wagner stated that Ms. Blackburn appeared to have a "history on exam consistent with Guillain Barré syndrome*." Id.* at 38.

Based on this GBS diagnosis, Dr. Wagner recommended that Ms. Blackburn be admitted to the Inpatient Neurology Service at the University Hospital for a course of intravenous immunoglobulin ("IVIG").[8] Pet'r's Ex. 12 at 38-39; Pet'r's Ex. 7 at 25, 29, 30. Dr. Wagner also

[7] AIDP refers to acute inflammatory demyelinating polyradiculoneuropathy, a common GBS variant. P. Dyck & P.K. Thomas, 2 *Peripheral Neuropathy* 2199 (4th ed. 2005) [hereinafter "Dyck & Thomas"].

[8] IVIG treatments are used with patients suffering from neuropathies like GBS and other illnesses believed to have an autoimmune character, and help patients maintain sufficient antibody levels. Dyck & Thomas at 642-43. The exact mechanism of action for IVIG in individuals with GBS is unknown, but the American Academy of Neurology Practice Parameter Group recommends "consideration of IVIg for patients within [two] weeks and probably within

ordered an EMG, which was performed on August 26, 2009. Pet'r's Ex. 3 at 45. Dr. Wagner's written interpretation of these EMG results stated that "[t]here is electrodiagnostic evidence of a severe demyelinating peripheral neuropathy consistent with the presumed diagnosis of Guillain Barré Syndrome (acute inflammatory demyelinating polyradiculoneuropathy, AIDP)." Pet'r's Ex. 5 at 124. Dr. Wagner's notes go on to indicate that "[t]he low compound muscle action potential amplitude in the median motor response implies significant axonal damages as to the fibrillations and positive waves in anterior tibialis muscle." *Id*.

During her hospitalization, Petitioner "received 2 g/kg of IVIG treatment over three days and developed nausea and vomiting, as well as anemia, which was diagnosed as hemolytic anemia" attributed to the IVIG treatment (not an uncommon occurrence).[9] Pet'r's Ex. 7 at 6-7. Despite such treatment complications, Ms. Blackburn completed her course of IVIG on August 28, 2009, showing "dramatic improvement in her symptoms*"* and was therefore discharged from inpatient neurology care at the University Hospital. *Id.* at 23, 25, 33. Dr. Wagner indicated upon discharge that Ms. Blackburn was to undergo physical rehabilitation for further treatment, and cautioned that "[g]iven the patient's reaction to Gardasil vaccine, she is not to complete the 3-step series and is also not to receive further vaccines given her risk of [GBS]." *Id*. at 24.

Petitioner was thereupon transferred to Acute Rehabilitation Service at the University of Utah for comprehensive treatment, as she was still experiencing difficulty in movement. Pet'r's Ex. 7 at 29-30. Petitioner nevertheless continued to be followed by the hospital's Neurology service. *Id.* at 19. A few weeks later, on September 5, 2009, Petitioner was discharged from her rehabilitation, having "made significant functional gains during her stay," with discharge diagnoses of "(1) [a]cute inflammatory demyelinating polyradiculoneuropathy; (2) [n]europathic pain; [and] (3) [h]emolytic anemia secondary to IVIG." *Id.* at 18, 20.

C. *Petitioner's Ongoing Symptoms and Eventual Change in Diagnosis*

Ms. Blackburn continued to undergo physical and occupational therapy throughout September of 2009. *See generally* Pet'r's Ex. 1. Despite her general progress and nascent recovery, results from a September 18, 2009 EMG study were interpreted by Dr. Wagner as abnormal due to "electrodiagnostic evidence of a primary demyelinating neuropathy with secondary axonal damage" as well as "evidence of early reinnervation."[10] Pet'r's Ex. 5 at 363.

[four] weeks of the onset of symptoms" even though "[a]ccording to a Cochrane systematic review, there are no adequate trials comparing IVIg to placebo" in patients with GBS. *Id.*

[9] Petitioner's medical records contain the observation that "[t]here are multiple case reports describing hemolysis in the setting of high-dose IVIG treatment." Pet'r's Ex. 7 at 27.

[10] Reinnervation is defined as the restoration, by regrowth or by grafting, of nerve supply to a part of the body from which it has been lost. Dyck & Thomas at 1421-22.

As of early October, Ms. Blackburn appeared to be recovering and reported a diminishment of her symptoms. Pet'r's Ex. 3 at 61. By October 22, 2009, however, when Ms. Blackburn was seen by Katarina Waters, PNP,[11] she had begun to experience increased bilateral numbness below her knees, weakness in bilateral lower extremities, and was also having trouble walking, making Petitioner concerned that she was suffering a relapse. Pet'r's Ex. 3 at 58. A physical examination revealed that her reflexes were (again) absent in both knees and ankles, and that she had decreased bilateral sensation from her knees down. *Id.* at 59. Because a neurologic evaluation could not be immediately scheduled, Ms. Blackburn was sent to the emergency room. *Id.* at 60.

At the hospital emergency department, Ms. Blackburn complained of "worsening weakness and numbness the onset of which had been gradual" but which had flared in the prior one and a half weeks. Pet'r's Ex. 3 at 39; Pet'r's Ex. 7 at 14-15. She reported that she had previously spoken to Dr. Rosenbluth about her symptoms and he indicated that she may need more IVIG, so she was told to come to the Emergency Department for possible admission. Pet'r's Ex. 7 at 15. A physical examination performed at this time revealed decreased sensation to touch of her lower extremities below her knee, no pain sensation of her feet, decreased temperature sensation to her bilateral feet, and loss of proprioception of her toes. Pet'r's Ex. 3 at 39-40. The doctors who evaluated her, however, did not conclude that inpatient admission for IVIG treatment was appropriate despite her symptoms. Pet'r's Ex. 7 at 16.

In the following days, Ms. Blackburn continued to experience the recurrence of many of the severe symptoms she had just received treatment for in August (*i.e.*, bilateral and ascending limb weakness), leading her to see Dr. Jackie J. Whitesell in the Neuromuscular Disease Clinic on October 29, 2009. Pet'r's Ex. 3 at 64-65. A physical examination confirmed the areflexia in her arms and legs (as observed at her emergency department visit earlier that month) as well as some numbness in her legs. *Id.* at 66. Dr. Whitesell indicated in her notes that "I feel that this likely still represents slowly improving Guillain-Barré which can sometimes have a waxing and waning course . . . I feel that an alternative diagnosis of CIPD [chronic inflammatory demyelinating polyneuropathy] is less likely." *Id*. Dr. Whitesell did not, however, explain why she discounted the possibility of CIDP, indicating only her conclusion that based on the examination, Petitioner had "AIDP with continued weakness and numbness in her extremities." *Id*.

Ms. Blackburn's symptoms did not dissipate (as had been anticipated when she first completed her IVIG treatment in late August 2009) in the months that followed. However, her condition did not impel her to visit another doctor again until the spring of 2010 – nine months after the vaccination. At that time, on April 22, 2010, Ms. Blackburn presented to Mark B. Bromberg, M.D. in the Neuromuscular Clinic at the University of Utah Hospitals and Clinic in

[11] Medical notes from that visit were electronically signed by Dr. Rosenbluth. Pet'r's Ex. 3 at 58.

Salt Lake City, Utah with complaints of limb weakness that had for the past six months fluctuated on an almost daily basis. Pet'r's Ex. 7 at 6-7. Although Petitioner reported that she was doing better, she indicated that she had not returned to baseline and was still experiencing "aches and pains, with neuropathic pain, as well as muscle spasms." *Id*.

Dr. Bromberg's notes state that Ms. Blackburn was "here for another evaluation for her history of AIDP with a question of if there is a component of chronic involvement or just delayed and incomplete recovery." Pet'r's Ex. 7 at 8. However, given Petitioner's history of unresolved, waxing-and-waning symptoms, Dr. Bromberg questioned whether the prior AIDP diagnosis was in fact correct. *Id.* As he specifically indicated:

> [h]er overall history does fit a monophasic illness, given the vaccine-related symptoms, as well as the lack of true exacerbation throughout her course in the past couple months. However, she still has significant disability from her arm and leg weakness, as well as pain. At this point, we would like to give her a trial of prednisone,[12] given that she has had more side effects with IVIG in the past. [] *We would expect significant and noticeable clinical benefit if this were CIDP and if she does not have significant response, then this steers us towards this being a monophasic AIDP with prominent axonal injury and delayed and perhaps incomplete recovery*.

Id. (emphasis added). Accordingly, Ms. Blackburn was prescribed prednisone and instructed to come in for a follow-up appointment within five to six weeks. *Id.* at 8-9.

By the time of Ms. Blackburn's follow-up visit on June 24, 2010, Dr. Bromberg was able to observe that since beginning prednisone two months prior, Petitioner exhibited "subjective and objective improvement." Pet'r's Ex. 7 at 2-3. He thus indicated in his notes that Petitioner "was *thought initially to have AIDP. However, given her lack of complete improvement and now improvement on prednisone, this suggests that she has rather CIDP as explanation of her symptoms*." *Id.* at 4 (emphasis added).

D. *Ms. Blackburn's More Recent Treatment History*

Since Ms. Blackburn's diagnosis was changed to CIDP, she has (consistent with that diagnosis) continued to experience symptoms on a waxing and waning basis, and has thus been seen regularly by a number of providers to follow-up on and attempt to manage her symptoms. But nothing in the subsequent record contradicts the consensus among Ms. Blackburn's treating physicians that the initial diagnosis of AIDP had been in error. *See, e.g.*, Pet'r's Ex. 12 at 6 (Dr. Bromberg's notes from her February 2011 visit). Consistent with that, any initial efforts to take Ms. Blackburn off prednisone lead to a recurrence of her symptoms such as numbness in her upper and lower extremities. *Id*.

[12] Prednisone is "a synthetic glucocorticoid derived from cortisone, administered orally as an anti-inflammatory and immunosuppressant in a wide variety of disorders." *Dorland's* at 1509.

In a May 2011 follow-up visit with Dr. Bromberg, it was noted that Petitioner was continuing to take prednisone and was doing well since her last visit other than experiencing some mild paresthesias in her limbs and fatigue – symptoms that Dr. Bromberg characterized as natural fluctuations commonly experienced by CIDP patients. Pet'r's Ex. 11 at 2-3. Petitioner thereafter continued to follow-up with Dr. Bromberg and other treatment providers to gauge her progress, and also to evaluate whether IVIG could ever be tried again in the event of another relapse (leading to a determination that future IVIG treatment was not contraindicated). Pet'r's Ex. 29 at 79-80, 85-88. Ms. Blackburn saw Dr. Bromberg again in the spring of 2012 to address the treatment management of her CIDP, and he concluded that her illness appeared to be in remission despite the tapering off of her steroid treatment. *Id.* at 77-78. The remaining medical records filed in this case indicate that Petitioner has not subsequently experienced a severe recurrence of her CIDP and has not required ongoing prednisone treatment.[13]

II. Expert Testimony

Three experts testified at hearing: one for Petitioner and two for Respondent. The qualifications and testimony of each side's respective experts are summarized below.

A. *Petitioner's Expert – Dr. Steinman*

Lawrence Steinman, M.D. is a professor in Stanford University's Departments of Neurology, Pediatrics, and Genetics, and the chair of Stanford's Immunology Program. Pet'r's Ex. 17 (Dr. Steinman's curriculum vitae). He has been elected to the Institute of Medicine ("IOM") and has published more than 400 articles, including articles related to his research on autoimmune disease and molecular mimicry. *Id.* Dr. Steinman's primary focus is on MS, but during the thirty-two years that he has been a board-certified neurologist he has treated approximately 400 patients with GBS (although, unlike Ms. Blackburn, approximately sixty-five percent of these patients that he has treated were children). Tr. at 144-46. Of those 400 patients, Dr. Steinman indicated that approximately twenty-five percent could be characterized as having CIDP. *Id.* at 146.

[13] On June 18, 2012, Petitioner presented for her six-month follow-up appointment. Pet'r's Ex. 29 at 7. Even without steroid treatment at that time, she generally appeared to be doing well, although she complained of some lingering upper and lower body bilateral weakness as well as occasional fevers the cause of which could not be identified by tests. *Id*. On November 26, 2012, Petitioner was again seen for a follow-up appointment and reported that she was doing well. *Id.* at 19. Thereafter, on April 18, 2013, Petitioner was seen for a follow-up appointment, and it was noted that she was "pregnant for 9 weeks now" and reported that she was doing well – although because she had decreased her doses of medication during her pregnancy, she was occasionally experiencing some of the old symptoms. *Id.* at 48-49.

Dr. Steinman admitted that due to his focus on research, he has seen considerably fewer patients in the last five years than he saw earlier in his career, but he still sees patients (including approximately eighty patients as of March 2014, which he estimated to be less than the number of patients that he saw in 2013). Tr. at 144-45. During the last five years, Dr. Steinman has only seen patients approximately one month out of the year when he serves as an inpatient attending physician, and during this time he has been called to diagnose an individual with GBS or CIDP no more than about sixteen or seventeen times (including once in 2014 as of March of that year). *Id.* at 147. Additionally, Dr. Steinman has not conducted nerve conduction studies or interpreted the results from such studies since his initial training. *Id.* at 149. Moreover, Dr. Steinman acknowledged that no patients with CIDP have been referred to him for management of their long-term care in the last five years. *Id.* at 147.

Dr. Steinman offered an opinion on two topics: (1) a theory by which he proposed the HPV vaccine Ms. Blackburn received could have caused an autoimmune response leading to her purported GBS, and (2) whether, based upon his review of Ms. Blackburn's medical records, he believed her illness was AIDP or CIDP.

1. Dr. Steinman's Molecular Mimicry Theory – Dr. Steinman proposed the theory of molecular mimicry to explain how the HPV vaccine could cause GBS (including the AIDP variant of GBS). Molecular mimicry has been defined to be a "sequence and/or conformational homology between an exogenous agent (foreign antigen) and self-antigen leading to the development of tissue damage and clinical disease from antibodies and T cells directed initially against the exogenous agent that also react against self-antigen." Institute of Medicine, *Adverse Effects of Vaccines: Evidence and Causality* at 70 (Stratton K. et al., eds. 2011) [hereinafter "Adverse Effects of Vaccines"]; *see also* Tr. at 21-24; Steinman L., *Autoimmune Disease*, 269 Scientific America 106-14 (Sept. 1993) (Pet'r's Ex. 19). As Dr. Steinman explained in his testimony, invading microbes mimic the structure of host proteins. Tr. at 21. Because of these shared structures, when certain hosts develop an immune response to the invading microbes, their immune systems confuse self with foreign proteins, attacking both. *Id.* at 22-23. For molecular mimicry to occur, sufficient homology (a term Dr. Steinman defined broadly as "similarity" (*Id.* at 36)) must exist between self and foreign antigens. *Id.* at 35, 38, 192.[14]

All forms of GBS, Dr. Steinman testified, are autoimmune diseases occurring after the body mounts an immune response to a foreign agent that accidently targets the body's own nerve tissue – through molecular mimicry. Tr. at 19. One of the most well-known examples of molecular mimicry as the mechanism for GBS, he stated, involves a structure shared between a

[14] Though homology is necessary to trigger molecular mimicry, Dr. Steinman conceded that the existence of homology alone does not lead automatically to the conclusion that molecular mimicry has or will occur. Tr. at 56-57. Indeed, homology occurs frequently without any clinical effect. Adverse Effects of Vaccines at 71. Most cross-reactivity does not even lead to autoimmunity. Tr. at 161-62.

bacterium called *Campylobacter jejuni* and gangliosides, sugar structures found on the surface of myelin (which is present in large quantities in human peripheral nerve tissue). *Id*. at 25. Thus, an antecedent *Campylobacter jejuni* infection can, through molecular mimicry, lead to a particular GBS variant called acute motor axonal neuropathy ("AMAN").[15] *Id.* Dr. Steinman opined that it is medically accepted (while not fully understood) that certain vaccines can also cause GBS,[16] initiating an autoimmune process the same way another microbe (like the *Campylobacter* bacterium) might. *Id*. at 35. The components of the wild viruses contained in many vaccines actually share molecular homologies with myelin structures present in the human body, and thus could produce the same reaction as a wild virus alone. *Id.*

Dr. Steinman proposed a specific means by which molecular mimicry occurs within an autoimmune reaction between the HPV vaccine and the body. Dr. Steinman contended that the HPV virus (portions of which the Gardasil vaccine contains[17]) shares "molecular similarities" with certain amino acid peptides making up myelin basic protein ("MBP"), which he defined as "the most common protein in central and peripheral nervous system myelin." Tr. at 29, 39-40. After receipt of the HPV vaccine, in the course of the human body's adaptive immune response the host's immune system would be "tricked" into attacking MBP, the putative target antigen of the autoimmune response. *Id.* at 51, 162, 192-93.

Dr. Steinman's conception of homology was specifically based on structural similarities between components of the HPV vaccine and host tissues, and he applied that paradigm to explain how MBP would be putatively attacked. Tr. at 36, 38. In his testimony, he repeatedly employed the metaphor of a "catcher's mitt" (a groove formed on the host tissues by the human leukocyte antigen ("HLA"))[18] holding a peptide from the vaccine which would then be presented to a host T cell.[19] *Id.* at 18-22, 156-57. The peptide would "fit" into the catcher's mitt due to its

[15] AMAN is an autoimmune neuropathy that principally targets motor nerve axons. Dyck & Thomas at 575; Tr. at 16. Because there is a much higher incidence of *Campylobacter jejuni* infection in China than the United States, AMAN is rarer here. Tr. at 34-35.

[16] Dr. Steinman specifically cited the epidemiologic evidence derived from the 1976 swine flu outbreak (as outlined in the Schonberger article (Schonberger, L.B. et al., *Guillain-Barré syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110(2) Am. J. Epidemiol. 105–23 (1979) (Pet'r's Ex. 25) and the Langmuir article (Langmuir, A.D. et al., *An Epidemiological and Clinical Evaluation of Guillain Barre Syndrome Reported in Association with the Administration of the Swine Influenza Vaccine*, 119 Am. J. Epidemiol. 841-79 (1984) (Pet'r's Ex. 26)) to support this proposition. Pet'r's Ex. 19 at 13.

[17] Gardasil is a recombinant (as opposed to live virus) quadrivalent vaccine prepared from virus-like particles of the major capside (L1) protein of HPV Types 6, 11, 16, and 18. Centers for Disease Control and Prevention, *Quadrivalent Human Papillomavirus Vaccine: Recommendations of the Advisory Committee on Immunization Practices (ACIP)*, 56(RR-2) MMWR 8 (2007) (Pet'r's Ex. 38 at 3, 10-11); Tr. at 40-42.

[18] HLA or "human leukocyte antigens" are "histocompatibility antigens governed by genes of the HLA complex (the human major histocompatibility complex [MHC])." *Dorland's* at 105.

[19] T cells are lymphocytes carrying their own unique receptors, some of which recognize the foreign peptide, while others recognize the self MHC molecule. Dyck & Thomas at 560-61.

structural homology with the host protein. *Id.* at 21-22. It would thereupon give instructions to the T cell, which would in turn migrate to MBP on the host's peripheral nerves and attack, causing the demyelination that characterizes GBS. *Id.* at 17, 155-57, 160-62.

Dr. Steinman contended that the molecular mimicry process by which MBP was attacked could involve T cells, antibodies produced from B cells, or both, with neither being predominant. Tr. at 17-18, 24, 155, 161.[20] However, although T cells and B cells recognize similar antigens, they play different roles in the adaptive immune system; T cells can only recognize structures that are bound to the HLA, while antibodies are not so constrained. *Id.* at 17, 24-25. Dr. Steinman's theory ultimately relied on T cells as playing the primary role in the destruction of myelin. *Id.* at 156-58.

In support of his assertion that MBP was the target antigen for the molecular mimicry process he outlined, Dr. Steinman relied upon the Cornblath study (David R. Cornblath et al., *Immunoreactive Myelin Basic Protein in Cerebrospinal Fluid of Patients with Peripheral Neuropathies*, 20 Annals of Neurol. 370 (1986) (Pet'r Ex. 32)). Tr. at 162. The Cornblath study found increased MBP in the spinal fluid of patients with GBS, although it did not find that MBP was the target antigen in GBS. Pet'r's Ex. 32 at 3741. He also relied heavily on an article – Kai W. Wucherpfennig et al., *Recognition of the Immunodominant Myelin Basic Protein Peptide by Autoantibodies and HLA-DR2-Restricted T Cell Clones from Multiple Sclerosis Patients*, 100 J. Clinical Investigation 1114 (1997) (Pet'r's Ex. 18) [hereinafter "Wucherpfennig"] – which studied cross-reactivity between MBP peptides, on the one hand, and antibodies and T cells, on the other, in multiple sclerosis patients. *Id.* at 28-29, 37.

Dr. Steinman next attempted to identify the precise homology involved in the purported cross-reaction between an epitope[21] of MBP and the components of the HPV vaccine. Tr. at 48, 55-56, 155. Relying on two laboratory studies in which an autoimmune disease (experimental autoimmune encephalomyelitis ("EAE"))[22] has been induced in mice using peptides from HPV 40, 32, or the HPV L2 protein[23], Dr. Steinman specifically identified HFFKN[24] as the critical

---

[20] Indeed, not only did Dr. Steinman not substantially distinguish the structural requirements for T cell and B cell mimicry, but at some points he conflated the two despite the differences in the way each functions. *See* Tr. at 17, 46.

[21] An epitope is the "antigenic determinant." *Dorland's* at 637. "[O]nly the portion of the protein or polysaccharide molecule known as the antigenic determinant (q.v.) combines with antibody or specific receptor on a lymphocyte." *Id.* at 103.

[22] EAE is an experimental model for MS. Steinman, L., *Autoimmune Disease*, 269 Scientific American 109 (1993) (Pet'r's Ex. 19).

[23] *See* Ruiz P.J. et al., *Microbial Epitopes Act as Altered Peptide Ligands to Prevent EAE*, 189 J. of Experimental Medicine 1275-84 (1999) (Pet'r's Ex. 21) [hereinafter "Ruiz"]; Ufret-Vicenty et al., *In Vivo Survival of Antigen Specific T Cells that Induce EAE*, 188 J. of Experimental Medicine 1725-38 (1998) (Pet'r's Ex. 23) [hereinafter "Ufret-Vicenty"].

sequence sharing homology with these HPV strains. *Id*. at 55-57, 168. Within this sequence, FKN is the "core motif" for MBP mimicry, but (based on Dr. Steinman's view of the structural character of homology) an FK or FKN sequence would be sufficient for the cross-reactive process to occur. *Id.* at 48, 168, 192-93; *see also* Pet'r's Mem. at 6.

As Dr. Steinman admitted on the witness stand, however, the homology analysis set forth in his written expert report was premised upon an error.[25] Tr. at 47. Dr. Steinman's report had described homology between MBP and the L2 protein of HPV (strains 7, 13, and 40). *Id*. at 48-49; Pet'r's Ex. 16 at 8. However, Gardasil is manufactured from the L1 protein of HPV (strains 6, 11, 16, and 18) – not the L2. Pet'r's Ex. 38 at 8. The sequences he described are thus not included as components of the Gardasil HPV vaccine, and therefore (as Dr. Steinman admitted) his initial analysis was "not germane" to the actual formulation of the relevant HPV vaccine. Tr. at 48-49, 135.[26]

Dr. Steinman attempted to correct his mistake through his live testimony, supported by additional medical literature filed before the March hearing. Tr. at 48-49**;** Pet'r's Ex's 41-44 (medical literature); Pet'r's Pre-Hr'g Reply Mem. (ECF No. 60) at 6-10; Pet'r's Ex. 46 at 1 (demonstrative exhibit). To do so, he indicated that after he discovered the error, he conducted what he termed "an experiment done, if you will, in silico [meaning performed on a computer] at my desk with publicly available databases." Tr. at 47. In effect, he looked up on the Internet the published genome sequences relevant to the actual components of the Gardasil vaccine and then compared them to the protein sequences found in MBP.

In so doing, Dr. Steinman determined that the L1 proteins contained in the Gardasil HPV vaccine included the amino acid sequences "YKN" and "FK."[27] Tr. at 52. Dr. Steinman then asserted that F and Y are "pretty much interchangeable," due to their shape and spatial position in the overall protein sequence, illustrating his point with visual charts representing how the

---

[24] "H" refers to the amino acid histidine, "F" refers to phenylalanine, "K" refers to lysine, and "N" refers to asparagine. Tr. at 39-44; Pet'r's Ex. 46 at 1. The amino acids in this sequence may be referred to as H88, F89, F90, K91, and N92. Wucherpfennig at 1117.

[25] Dr. Steinman's written report was filed in July 2012. ECF No. 36. Respondent pointed out in her January 28, 2014 pre-hearing memorandum (ECF No. 62), however, that Dr. Steinman had not compared the proteins actually contained in the relevant formulation of the HPV vaccine in this case to MBP. Because the hearing was held in late March of 2014, Petitioner had ample forewarning of the need to respond to this criticism of Dr. Steinman's reasoning, and did so during Dr. Steinman's testimony.

[26] Dr. Steinman attributed this error to the fact that when he wrote his report, he "stuck to the viruses that we had addressed in this paper by Wucherpfennig." Tr. at 48.

[27] "Y" refers to the amino acid tyrosine. Tr. at 43.

sequences appear in three dimensions after magnification. Tr. at 47, 43, 52-54.[28] From a homology standpoint, both "keys" would still fit the "lock" represented by the MBP peptide sequences, thereby inducing an autoimmune response. Accordingly, Dr. Steinman testified that he was still able to maintain the opinion that there is sufficient homology between the actual HPV vaccine received by Ms. Blackburn and MBP. *Id.* at 135, 192-93. Indeed – it was his opinion that the homology was even stronger than before, because now there were two possible sequences – FK and YKN – to provide a basis for molecular mimicry. *Id.* at 52, 143.

2. Dr. Steinman's Diagnostic Opinion – Dr. Steinman also offered an opinion on the nature of Ms. Blackburn's illness based on a review of her treatment history. Dr. Steinman challenged the concept that when Ms. Blackburn presented to Dr. Zimmerman between December of 2008 and January of 2009, her symptoms were consistent with the expected presentation of an individual suffering from CIDP. Tr. at 70, 92-96,123, 130-31. If an individual were suffering from CIDP, he reasoned, a treating physician would expect to observe numerous abnormalities from their neurologic exam. *Id*. at 92. However, Dr. Steinman pointed out that diagnostic testing, such as her December 2008 MRI, displayed essentially normal results,[29] or were logically consistent with the physical demands of her job. *Id.* at 88-89. He also observed that she displayed asymmetric pain and no areflexia.[30] *Id.* at 89-92. And the typical patient with CIDP, Dr. Steinman opined, would not be able to continue to work two jobs (one in the nursing profession and the other in housecleaning) without seeking treatment, yet Ms. Blackburn had done so. *Id.* at 70; 93-94. In the same vein, Dr. Steinman also noted the gap in Ms. Blackburn's medical records from January of 2009 to July of 2009 – a long time, in his view, for an individual with CIDP to not seek medical treatment. *Id.* at 90-92.

Dr. Steinman further testified that he considered the post-vaccination AIDP diagnosis to be especially reliable and improperly revised to CIDP in 2010. Tr. at 74-75, 123. For example, Ms. Blackburn's neurologist, Dr. Zimmerman, first observed areflexia on August 11, 2009, but not before. *Id*. at 95. In Dr. Steinman's reading of the records, this was a significant presenting symptom, and the fact that it was not seen before vaccination was very important. *Id.* at 96. He

[28] In support, Petitioner offered a blown-up x-ray diffraction photograph of the relevant portion of MBP in order to illustrate precisely where on the HLA "catcher's mitt" the homologous portion of the HPV vaccine peptide would bind by showing the shapes or structure of the relevant peptide sequences. Tr. at 39-40, 42-44 (referencing Pet'r's Ex. 46).

[29] In addition Ms. Blackburn's cranial/cerebellar/sensory motor responses and reflexes (as tested in December 2008 and January 2009) were within normal limits. Tr. at 91-92. Dr. Steinman further observed that Ms. Blackburn had normal reflexes with intact sensation, which he opined was not supportive of a diagnosis of ongoing inflammatory demyelination. *Id.* at 87-88.

[30] Ms. Blackburn was experiencing shooting pain down one leg, which in Dr. Steinman's view was not consistent with an ongoing process of CIDP where pain would normally be symmetrical. Tr. at 89. Nor did he consider the physical therapy treatment prescribed for Petitioner at that time normal treatment recommended for a patient with CIDP. *Id.* at 89-90.

also considered it important that her subsequent lumbar puncture revealed the anticipated albumin dissociation that is a clinical hallmark of GBS. *Id.* at 105. He questioned whether certain symptoms that Respondent argues are critical for a GBS diagnosis that were absent (such as cranial facial muscle weakness, respiratory weakness, and autonomic instability) were in fact critical. *Id.* at 71. Dr. Steinman deemed particularly significant the abnormal results obtained from the EMG performed on September 18, 2009, which revealed secondary axonal damage[31] – something he considered uncommon in the case of CIDP. *Id*. at 66, 112-13.[32]

Dr. Steinman also stressed those instances in the treating record where Ms. Blackburn's physicians linked the Gardasil vaccination to her GBS. Tr. at 108. He discounted the possibility that Ms. Blackburn's treating physicians had not taken her prior medical history into account in so concluding. *Id.* at 109. He admitted, however, that to some extent his conclusion arose from his assumption that in a "teaching hospital" such as where Ms. Blackburn was treated any diagnosis could be assumed to have taken into account a patient's medical history. *Id.* at 109-10.[33]

Dr. Steinman explained the waxing and waning quality of Ms. Blackburn's symptoms after her hospitalization as the product of an incomplete recovery from her initially acute GBS. He attributed some of her relapse (which he admitted was "pretty big" (Tr. at 116)) to her having received an incomplete series of IVIG treatments due to the hemolytic anemia she developed. *Id.* at 115. This new phase of her disease thus did not negate the AIDP diagnosis, as recurrence is not unknown to AIDP patients. *Id.* at 115-16, 119. Dr. Steinman also emphasized Ms. Blackburn's neurology consult with Dr. Whitesell (who rejected a CIDP diagnosis), and the subsequent follow-up EMG and nerve conduction studies, the results of which confirmed Petitioner's recovery (showing reinnervation). *Id*. at 118-120. Had Ms. Blackburn been suffering

[31] As Dr. Steinman explained, secondary axonal damage refers to the fact that after the overlying myelin sheath gets damaged, the axon within can also become damaged from the consequences of the initial process. Tr. at 112. He noted that the AMAN form of GBS is more commonly associated with secondary axonal damage, but distinguished Ms. Blackburn's case by observing that motor and sensory axons were involved (which would not be true for AMAN). *Id.* at 112-13.

[32] In support of this part of his opinion, Dr. Steinman referenced an article (L. Ruts, M.D. et al., *Distinguishing Acute-Onset CIDP From Fluctuating Guillain-Barré Syndrome*, 74 Neurol. 1680-86 (2010) (Resp't's Ex. H)) citing a study of GBS and CIDP which found signs of axonal damage were "rare in acute onset CIDP group, while more than half of the patients in the GBS treatment-related fluctuations group show signs of axonal damage in the acute phase." Tr. at 66.

[33] Dr. Steinman testified that in his experience, a teaching hospital (such as the University of Utah where Ms. Blackburn received care) is typically staffed with extremely bright interns, residents, and faculty members who are challenging every diagnosis. Tr. at 74. Accordingly, Dr. Steinman opined that "for the doctors and professors at a major university teaching hospital to have made the diagnosis of acute inflammatory demyelinating polyneuropathy based on clinical presentation, spinal fluid examination, electrophysiology and all the challenges that go on from the team, to me, chances are [ninety-nine] percent that that diagnosis was correct." *Id.* at 75.

from ongoing CIDP, Dr. Steinman testified, he would have expected to see the opposite (less reinnervation, and more dennervation). *Id.* at 120-21.

Dr. Steinman specifically disputed the accuracy of changing Ms. Blackburn's diagnosis to CIDP. He believed the improvement in her overall health by the spring of 2010 supported the conclusion that she merely suffered from a waxing and waning form of AIDP. Tr. at 124. He further noted that in his experience, AIDP patients often had incomplete recoveries – and that therefore this was an insufficient reason to alter a patient's initial diagnosis. *Id.* at 62-63, 127-28, 131. Dr. Steinman went on to observe that reflexes were present during this visit and continued to be present at every bodily site (including her ankles) at subsequent visits in the months to follow. *Id.* at 128-30. He admitted, however, that based on the very same treatment history, many of his colleagues would likely change a patient's diagnosis to CIDP. *Id.* at 62-63.

Finally, Dr. Steinman questioned the significance of Ms. Blackburn's responsiveness to prednisone, asserting that steroids such as prednisone would inherently tend to make a patient feel less depressed and even "ebullient." Tr. at 126-28. He added that he was not in favor of prednisone as a treatment due to its "nasty side effects." *Id.* at 130. He later admitted, however, that prednisone was in fact indicated for CIDP[34], but asserted that it represented a disfavored, last-ditch treatment when more standard treatments like IVIG had failed. *Id.* at 181-83. At bottom, Dr. Steinman felt that the mere temporal coincidence of corticosteroid treatment and continued recovery from a demyelinating episode was insufficient to establish CIDP as the correct diagnosis. *Id.* at 72.

B. *Respondent's Experts*

1. Dr. Chaudhry – Respondent's expert, Vinay Chaudhry, M.D., received his medical degree from All-India Institute of Medical Services in New Delhi, India. Tr. at 194; Resp't's Ex. B (Dr. Chaudhry's curriculum vitae). He then went to England for secondary training where he obtained Membership in the Royal College of Physicians (M.R.C.P). Tr. at 194. Thereafter, Dr. Chaudhry completed a residency in neurology at the University of Tennessee Center for the Health Sciences and the University of Alabama in Birmingham, followed by a two-year electromyography ("EMG") and neuromuscular disease fellowship training program at Johns Hopkins University. *Id.* Dr. Chaudhry joined the faculty at John Hopkins in 1989, where he currently works as a professor of neurology, teaching and working on

[34] Dr. Steinman also attacked Dr. Chaudhry's contention that steroids are never to be used for treatment of GBS, arguing instead that they were in fact commonly used until it became clear that IVIG and plasmapheresis treatments were more successful. Tr. at 73-74.

clinical trials.[35] *Id*. at 194, 196. He is board-certified in clinical neurophysiology, neuromuscular medicine, and electrodiagnostic medicine. *Id.* at 199. He has also published articles on a variety of topics, including GBS, with most of his publications pertaining to some aspect of peripheral neuropathy. *Id.*

Despite his academic duties, Dr. Chaudhry spends the majority of his time caring for patients, including patients suffering from GBS, CIDP, and other neuromuscular diseases. Tr. at 196-97. Most of Dr. Chaudhry's outpatients suffer from CIDP, and he assists such individuals with diagnosis and management of long-term care. *Id.* at 196-98. Patients with GBS, by contrast, are typically so acutely ill that they require inpatient hospitalization (and then rarely return to the hospital thereafter because they have recovered), but one month a year Dr. Chaudhry sees GBS patients as well in conjunction with his work as an attending physician. *Id.* at 169, 208. Dr. Chaudhry also sees GBS patients for diagnostic purposes in the EMG laboratory at Johns Hopkins (where his responsibilities include acting as co-director of the laboratory, which is responsible for conducting about 5,000 of such studies per year), and he is directly experienced in conducting such tests and interpreting their results. *Id.* at 196-98, 218-19. Dr. Chaudhry estimated that over the course of his twenty-five year career, he has seen close to 30,000 patients with either GBS or CIDP. *Id.* at 197.

Consistent with the opinions expressed in his written reports (*see* Resp't's Ex. A at 5; Resp't's Ex. XX at 2), Dr. Chaudhry testified that Petitioner's CIDP diagnosis was correct.[36] Tr. at 201-02, 209-41; *See also* Resp't's Ex's E-J (medical or scientific literature relied upon by Dr. Chaudhry in formulating this opinion). While acknowledging that CIDP is a difficult diagnosis to make, even under the best circumstances (Tr. at 197-98), Dr. Chaudhry expressed the opinion that this is the diagnosis that all of Ms. Blackburn's providers would have eventually made – even those who initially surmised that she suffered from AIDP – had they had the benefit of reviewing the totality of her medical records (including the clinical, electrophysiological, laboratory, and therapeutic features of her disease). *Id.* at 222-23, 249-51.

Dr. Chaudhry reached his conclusion that Petitioner had CIDP largely based on the evidence of waxing and waning of her symptoms over a large period of time (including before her receipt of the HPV vaccination). Tr. at 239-41. Thus, he viewed the symptoms Ms. Blackburn exhibited in late 2008 as initial evidence of her CIDP. *Id.* at 210. Indeed, Ms.

[35] Dr. Chaudhry's involvement in clinical research includes serving as an investigator on research trials; for example, he was involved in the clinical trials that recently lead the FDA to approve IVIG for treatment of CIDP. Tr. at 200.

[36] Dr. Chaudhry also opined that, given the diagnosis and the onset of symptoms that preceded the vaccination, he did not believe that the HPV vaccine played a significant role in Ms. Blackburn's alleged injury. Tr. at 202. My decision does not, however, rely on this portion of his testimony (which was in fact mooted by Dr. Chaudhry's testimony about the pre-vaccination course of Ms. Blackburn's CIDP).

Blackburn's initial symptoms were referred to as neuropathic or neuralgic several times in the contemporaneous medical records, and she was prescribed medications typically given to patients who are experiencing neuropathic or neuralgic conditions. *Id.* Dr. Chaudhry also found it significant that Dr. Zimmerman suggested performing tests aimed specifically at ruling out nerve injury, nerve damage, or neuropathy of some sort, such as nerve conduction studies.[37] *Id*. at 228. And that Mr. Blackburn reported experiencing tingling in her legs and feet on the date of her July 2009 vaccination (at which time she was still taking medication for her prior neuropathic symptoms). *Id.* at 211-12**.** Based on all of the above, Dr. Chaudhry concluded that Petitioner's CIDP had likely begun in the second half of 2008. *Id.* at 227, 249-50.

Dr. Chaudhry found further support for his opinion that Ms. Blackburn's CIDP predated her vaccination in Petitioner's August 19, 2009 EMG and nerve conduction study results (based on tests performed within a month of her vaccination). Tr. at 216-18. Having conducting hundreds of such tests over the years, Dr. Chaudhry indicated that he would expect a typical GBS patient to have EMG results displaying relative preservation (sparing) of sensory nerve action potentials. *Id.* at 216. Ms. Blackburn's test results, however, showed no response in any of her sensory nerves. *Id.* He also observed reduced recruitment in her EMG results,[38] demonstrating to him that Ms. Blackburn already had a severe demyelinating neuropathy. *Id.* at 217. Assuming Ms. Blackburn's GBS began as a result of her July 2009 vaccination, it would be highly unusual for a patient to display such extensive damage in so short a time, whereas it would be common for CIDP (which would have progressed over a much greater length of time and begun many months before). *Id.* at 216, 218.

On the other hand, Dr. Chaudhry opined that Ms. Blackburn did not display post-vaccination the kind of symptoms that he believed were consistent with a GBS diagnosis. Tr. at 220. For instance, Ms. Blackburn presented with complaints of three-and-a-half weeks of ascending weakness and numbness on August 24, 2009, but when examined, Ms. Blackburn did not exhibit any facial weakness – her cranial facial nerve appeared normal. *Id*. at 219-22. Her upper extremities were also close to normal, which in Dr. Chaudhry's view would be unusual for someone suffering from an acute disease like GBS that purportedly began about a month earlier (and should be at its most severe point by that time). *Id.* at 219-21.[39] And he questioned Dr.

[37] Even if her symptoms were not severe enough at this point for the doctor to order a lumbar puncture or nerve conduction studies, they still suggested to Dr. Zimmerman that he do preliminary testing, and start treating Ms. Blackburn with amitriptyline. Tr. at 226-28.

[38] "In a normal recruitment pattern, the number of discharging motor units increases appropriately for the muscle force generated by the effort. In neuropathy, a loss of functional motor units results in late or decreased recruitment associated with rapid firing of discharging units to compensate for the reduced number." Dyck & Thomas at 952-53 (citations omitted).

[39] Based on his review of the contemporaneous records and test results from Ms. Blackburn's August 24th doctor's visit, Dr. Chaudhry actually concluded that even if he accepted the GBS diagnosis, the time from onset at this point

Steinman's belief that Ms. Blackburn's post-hospitalization relapse was attributable to her not receiving a full course of IVIG treatment, since (based on the medical records) it appeared to Dr. Chaudhry that she had received a full course of treatment despite her anemic reaction. *Id.* at 224-25, 233-34.[40]

The success of the prednisone course that Dr. Bromberg prescribed was in Dr. Chaudhry's view particularly strong evidence that CIDP was the correct diagnosis. Tr. at 235-36, 238-39. In discussing the benefit of prednisone for CIDP patients, Dr. Chaudhry took issue with Dr. Steinman's suggestion that Ms. Blackburn felt better after receiving it simply because of its "euphoric effect." *Id.* at 239. Dr. Chaudhry emphasized that Ms. Blackburn's intervening medical history corroborated the benefit of the prednisone treatment, since her symptoms got worse whenever her dosage was removed or reduced. *Id.* In fact, when Dr. Bromberg began a slow taper of the prednisone, he started Ms. Blackburn on CellCept[41] – a very strong steroid sparing agent that (in Dr. Chaudhry's experience) would never be given to a patient believed to have GBS. *Id.* at 237. Nothing Dr. Chaudhry saw in Ms. Blackburn's medical history suggested to him that the diagnosis change was in error, nor did any of her subsequent treating physicians believe that CIDP was a mistaken diagnosis in light of additional findings or data pertaining to her progress. *Id.* at 239-40, 249-50.

Dr. Chaudhry addressed some of Petitioner's arguments that Ms. Blackburn did not display any CIDP symptoms pre-vaccination. He acknowledged that Ms. Blackburn's reflexes were recorded as normal when she received the vaccination as well as before, but opined that her other symptoms, viewed together, likely represented the beginning of a neuropathy. Tr. at 212-13. In his experience, however, it was typical for CIDP patients to experience a variety of mild neuropathic symptoms over a long period of time before receiving a CIDP diagnosis, despite the presence of normal reflexes.[42] *Id*.

He did not find significant the long gap between Ms. Blackburn's early 2009 doctor's visits and the post-vaccination flare-up she experienced, since in his view it was common for

---

would have to have been longer than four weeks and closer to sixty days (six weeks or thereabouts), which is well outside the clinical definition for the disease's four-week progression. Tr. at 221-22.

[40] *See* Pet'r's Ex. 7 at 25; Tr. at 224. Dr. Chaudhry's understanding of what a full IVIF course would be is consistent with prominent medical literature on the subject. *See, e.g.*, Dyck & Thomas at 642 (the standard course of IVIg "first used for autoimmune thrombocytopenia was 0.4g/kg daily for [five] days, and this has been adopted in neuromuscular diseases" (although the dose may be different for recurrent courses)).

[41] CellCept (mycophenolate mofetil) is an immunosuppressive agent (*Dorland's* at 1216) more commonly used for renal transplant patients. Tr. at 237.

[42] Dr. Chaudhry speculated that Ms. Blackburn's initial treatment providers may not have been as comprehensive in testing her reflexes (given the other warning signs present) as he would have given his experience. Tr. at 213.

CIDP patients to go a long time with low grade or intermittent symptoms while still being able to work. *Id.* at 206, 226. Dr. Chaudhry found it unlikely that a twenty-six year old would experience this type of nerve pain and tingling as the result of mere "physical stress" (Resp't's Ex. A at 6-7; Tr. at 227), and pointed to Dr. Zimmerman's suspicion of neuritis or neuralgia as supportive of this view. Resp't's Ex. A at 6; Tr. at 228.

2. Dr. Whitton - James Lindsay Whitton, M.D. is an immunologist at Scripps, a nonprofit private research institution. Tr. at 269; Resp't's Ex. D (Dr. Whitton's curriculum vitae). He serves on the editorial boards of several medical journals. Tr. at 272. Dr. Whitton has "only worked a little in molecular mimicry," though he has had professional colleagues with greater expertise in the topic. *Id.* at 269, 362. Dr. Whitton's testimony was offered in response to Dr. Steinman's proposed theory by which the HPV vaccine could cause GBS. Dr. Whitton proposed that Dr. Steinman's theory relied upon two core premises: (1) homology between the protein components of the HPV vaccine and the relevant target sites on the peripheral nerves; and (2) the induction of an immune response against MBP. *Id*. at 278-79. Dr. Whitton's disputed both premises, arguing that (1) MBP is not the target antigen in GBS; and (2) Dr. Steinman's homology theory is not scientifically valid.

Dr. Whitton agreed with Dr. Steinman that molecular mimicry is a biologically plausible theory under certain circumstances. Tr. at 362-63. He similarly did not challenge Dr. Steinman's general explanation for how molecular mimicry would occur. *Id.* at 281, 362. And he did not seriously question the assertion that GBS could be caused by molecular mimicry. *See* Resp't Ex. C (Dr. Whitton's Expert Report) at 4. Instead, Dr. Whitton challenged Dr. Steinman's invocation of molecular mimicry in this particular case. Tr. at 306.

First, Dr. Whitton questioned the application of Dr. Steinman's homology theory to the autoimmune response specifically resulting in GBS. Tr. at 295. Dr. Whitton explained that peripheral nervous system diseases are generally thought to be driven by antibody responses rather than T cell mediated. *Id.* at 327. He acknowledged the literature establishing that MBP-specific T cells induce EAE in mice via molecular mimicry (*Id.* at 283-86), but asserted that such studies were of limited applicability here, as they involved an artificial stimulation of animal immune systems intended to produce a *central* nervous system disease that nevertheless did not also cause the test subject laboratory animals to develop peripheral nerve diseases. *Id.* at 281-83. In Dr. Whitton's view, the "pathology and parthogenesis" of central nervous system neuropathies could not be properly conflated with peripheral nervous system neuropathies. *Id.* at 283.

Importantly, Dr. Whitton argued, the MBP-specific T cells that have been shown in the EAE studies to target MBP have not been shown to have the same effect in causing peripheral nervous system diseases. *Id.* at 286. This, he elaborated, was due to the fact that there is no evidence that MBP is a possible target antigen in causing GBS. *Id.* at 287. Rather, he noted that

there are many other potential structures in peripheral myelin (of which MBP is a component) that could be the target of a demyelinating autoimmune response. *Id.* at 291. In particular, nerve gangliosides[43] have been demonstrated to be the target in autoimmune-induced GBS via the process of molecular mimicry between the infectious agent and the ganglioside. *Id.* at 289-91; Tomoko Komagamine & Nobuhiro Yuki, *Ganglioside Mimicry as a Cause of Guillain-Barré Syndrome*, 5 CNS & Neurological Disorders 391-400 (2006) (Rep't's Ex. R at 6-7) [hereinafter "Komagamine"].

Second, Dr. Whitton questioned the scientific validity of Dr. Steinman's revised theory of homology. Dr. Whitton asserted that F and Y are in fact not generally interchangeable. Tr. at 310. Moreover, Dr. Whitton argued that there is "nothing special about FK" (Tr. at 317), pointing out that the probability that a protein of 500 amino acids contains at least one FK sequence is about seventy-one percent. *Id.* at 314. Thus, according to Dr. Whitton, if, as Dr. Steinman proposed, a sequence of that short length were sufficiently homologous with MBP to induce an autoimmune response, then it would be reasonable to expect autoimmune diseases to be far more common than they actually are. *Id.* at 315-17.[44]

III. Medical Literature

Both sides offered substantial scientific and medical literature.[45] Petitioner submitted twenty-five articles relied upon by her expert in formulating an opinion in this case. This included ten articles initially relied upon by Dr. Steinman to provide a mechanistic explanation of how the HPV vaccine could cause neurological damage, and to support his opinion that Ms. Blackburn's GBS was caused by her receipt of the vaccination in question.

---

[43] The term ganglioside refers to any of a group of glycosphingolipids (ceramide and oligosaccharide) occurring in nervous system tissues. *Dorland's* at 760,794.

[44] Dr. Whitton also addressed whether it appeared from Ms. Blackburn's medical history that she had suffered from GBS as a result of the vaccine rather than an antecedent infection, and also whether the proposed theory by which the HPV vaccine had caused Ms. Blackburn's purported AIDP occurred in a medically-acceptable timeframe. *See generally* Tr. at 319-40. Because my decision turns on other aspects of Dr. Whitton's testimony, however, or matters that he did not specifically address (such as the propriety of the GBS diagnosis), I do not review these additional portions of his testimony herein.

[45] Although I do not reference or discuss in this decision every item of medical or scientific literature offered by the parties, I have read all of the submitted articles. *Hazlehurst v. Sec'y of Health & Human Servs.,* 604 F.3d 1343, 1352 (Fed. Cir. 2010) (indicating that on review it is not necessary to rely on the presumption that the finder of fact has reviewed all presented evidence unless he specifically states otherwise, where the opinion of the special master specifically refers to that evidence). *See also Andreu v. Sec'y of Health & Human Servs*., 569 F.3d 1367, 1379 (Fed. Cir. 2009) ("Although *Althen* and *Capizzano* make clear that a claimant need not produce medical literature or epidemiological evidence to establish causation under the Vaccine Act, where such evidence is submitted, the special master *can* consider it in reaching an informed judgment as to whether a particular vaccination likely caused a particular injury") (emphasis added).

Respondent cites certain pieces of literature submitted by Petitioner as well as forty-seven additional articles relied upon by her experts in formulating their opinion in this case. Respondent initially submitted six articles used to support Dr. Chaudhry's expert opinions, which consisted of literature addressing the possible competing diagnoses as well as whether the vaccination in question triggered Ms. Blackburn's alleged illness. Respondent then submitted one additional article regarding the molecular pathogenesis of GBS. Respondent also submitted forty articles relied upon by Dr. Whitton to address the timing of the first onset of Petitioner's neurological disease in relation to her receipt of vaccination, as well as the likelihood that Ms. Blackburn's neurological disease was triggered by a prior infection rather than the HPV vaccination.

IV. Procedural History

As noted above, Ms. Blackburn filed this petition in June 2010. Petition (ECF No. 1) at 2-3. Over the ensuing six months, she filed relevant medical records and an affidavit that she intended to rely upon to establish her entitlement to compensation, certifying completion of the record on January 13, 2011. ECF No. 17. Thereafter, on March 9, 2011, Respondent filed her Rule 4(c) Report (ECF No. 20 ("Resp't's Rep't")), asserting in it that Petitioner could not satisfy the test for establishing entitlement to a Vaccine Program award as set forth in *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005). Resp't's Rep't at 14. Among other things, Respondent argued that the medical records did not themselves establish causation because none of Petitioner's treating physicians provided a medical theory. *Id.* Respondent also proposed that onset occurred before the vaccination, and/or was related to an antecedent infection, and further questioned the accuracy of the GBS diagnosis. *Id.* at 15.

Ms. Blackburn subsequently filed additional medical records in support of her claim, and then filed a second statement of completion on August 21, 2011. ECF No. 25. A status report filed on that same date indicated that Petitioner's counsel was preparing a demand letter to send on Petitioner's behalf to Respondent with the aim of resolving this case through settlement. ECF No. 25. Such initial settlement efforts were unsuccessful, however.

Petitioner requested and was granted a number of extensions of time to file an expert report in support of vaccine causation[46] before she did so on July 30, 2012. ECF No. 36. On September 25, 2012, Respondent filed an unopposed motion requesting an extension of time to file her expert reports, which was subsequently granted. ECF Nos. 38-39. Thereafter, on November 27, 2012, Respondent filed two expert reports. ECF Nos. 40-45. The special master

[46] On October 31, 2011, January 30, 2012, March 30, 2012, and May 30, 2012, Petitioner requested and was subsequently granted requests for an extension of time for filing an expert report in support of vaccine causation. ECF Nos. 27-31, 34-35.

formerly responsible for this case next conducted a telephonic status conference on December 20, 2012, to discuss the merit of each party's position in this case. ECF No. 46. During the conference, the parties again expressed the desire to explore the possibility of resolving this case through informal settlement negotiations prior to setting a date for an entitlement hearing. *Id.* But these additional settlement discussions proved unsuccessful, and so during a status conference on June 6, 2013 the case was set for hearing and the parties were instructed to complete the filing of relevant medical records as well as pre-trial briefs. ECF Nos. 55, 58.

In early 2014, the parties filed some additional medical literature, prehearing memoranda, and other supplemental materials. Thereafter, in March of 2014 the matter was assigned to me (ECF Nos. 69-70) but the previously-scheduled hearing dates (March 25-26, 2014) were maintained and the matter was tried as scheduled. Both parties filed post-trial briefs in June (ECF Nos. 77-78) and then responded to each other's filings in July (ECF Nos. 79-80). This matter is now ripe for adjudication.

V. Standards for Entitlement Award in Vaccine Program Cases

To receive compensation under the Vaccine Program, a petitioner must prove either: (1) that she suffered a "Table Injury" – i.e., an injury falling within the Vaccine Injury Table – corresponding to one of the vaccinations in question, or (2) that her illnesses were actually caused by a vaccine (a "non-Table injury"). *See* §§ 300aa-13(a)(1)(A),11(c)(1); § 300aa-14(a), as amended by 42 C.F.R. § 100.3; 300aa-11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Human Servs*., 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006).[47] No table injury is alleged in this case, so Ms. Blackburn must prove causation-in-fact.

Petitioners bear the burden of demonstrating actual causation by preponderant evidence. *Cedillo v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); § 300aa-13(a)(1). To do so, a petitioner must provide: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278. The preponderance standard requires a petitioner to demonstrate that it is "more likely than not" that the vaccine at issue caused his injury. *Moberly*, 592 F.3d at 1322 n.2. Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs*., 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner

[47] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit decisions are binding on special masters. *Guillory v. Sec'y of Health & Human Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd*, 104 F. App'x 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Human Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Human Servs*., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). To determine if Petitioner has carried her burden, I must assess "the record as a whole" and may not make an entitlement decision in her favor based solely on her own claims "unsubstantiated by medical records or by medical opinion." § 13(a)(1).

Each of the *Althen* prongs requires a different showing (although the preponderant evidence standard applies to each, and the same evidence can be offered to prove more than one of the prongs). Under *Althen* prong one, petitioners must provide a "reputable medical theory" demonstrating that the vaccine *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citations omitted). To satisfy this prong, petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). The theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy *Althen* prong one without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or by offering a theory that has general acceptance in the medical or scientific communities. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325-26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Andreu*, 569 F.3d at 1380.

Often, however, establishing a sound and reliable medical theory requires that the parties present expert testimony in support of their claims. *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.,* 509 U.S. 579, 594-96 (1993). *Cedillo*, 617 F.3d at 1339 (citing *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). "The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592-95).

In other federal judicial fora (such as the district courts), the *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to resolve admissibility questions, excluding evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the scientific evidence actually proffered and heard. *Davis v. Sec'y of Health & Human Servs.*, 94 Fed. Cl. 53, 66-67 (Fed. Cl. 2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"), *aff'd*, 420 F. App'x 923 (Fed. Cir. 2011). The flexible use of the *Daubert* factors to determine the persuasiveness and/or reliability of expert testimony in Vaccine Program cases has routinely been upheld. *See, e.g.*, *Snyder v. Sec'y of Health & Human Servs.,* 88 Fed. Cl. 706, 742-45 (2009).[48]

Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339,1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325-26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Human Servs.,* 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. In evaluating whether this prong is satisfied, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu,* 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as trustworthy evidence, since they are created

[48] Petitioner argued at length in her pre-trial submissions that *Daubert* had been incorrectly cited by Respondent because it is a tool for determining only if evidence was admissible, and therefore has less utility in evaluating the reliability, persuasiveness, or adequacy of expert testimony. *See, e.g.,* Pet'r's Pre-Hr'g Reply Mem., dated February 18, 2014 (ECF No. 68) at 10-20. This is plainly against relevant controlling authority.

contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records setting forth a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. § 300aa–13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder,* 88 Fed. Cl. at 745 n.67 ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). Rather, as with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence present in the record – including conflicting opinions among the treating physicians themselves. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 742, 749 (Fed. Cl. 2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 119, 136 (Fed. Cl. 2011), *aff'd*, 463 F. App'x 932 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344.

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.*; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (Fed. Cl. 2011), *recons. den'd after remand,* 105 Fed. Cl. 353 (2012), *aff'd mem.,* 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877, at *27 (Fed. Cl. Spec. Mstr. May 30, 2013), *aff'd*, No. 2014-5054, 2014 WL 6804880 (Fed. Cir. Dec. 4, 2014).

The law pertaining to the parties' respective burdens of proof also merits brief mention. Petitioner has suggested that because Respondent argues that Ms. Blackburn actually suffered from CIDP rather than GBS, it is Respondent's burden to establish Petitioner's CIDP as a "factor unrelated" cause of Petitioner's illness. *See, e.g.*, Pet'r's Pre-Hr'g Reply Mem. (ECF No. 68) at 2 (citing *Walther v. Sec'y of Health & Human Servs.*, 485 F.3d 1146, 1152 (2007)). It is correct that in cases where a petitioner successfully satisfies her initial burden of proof, the burden then shifts to Respondent to establish (also by the same preponderance of the evidence standard) that the petitioner's injuries are due to "factors unrelated" to the vaccines. *C.K. v. Sec'y of Health &*

Human Servs., 113 Fed. Cl. 757, 766 (2013) (citing *Knudsen*, 35 F.3d at 547); *Deribeaux v. Sec'y of Health & Human Servs.,* 105 Fed. Cl. 583, 587 (2012), *aff'd*, 717 F.3d 1363 (Fed. Cir. 2013); 42 U.S.C. § 300aa–13(a)(1)(B).

The burden of proof does not, however, shift to Respondent merely because (as in this case) she disputes the factual nature of the alleged injury or its proper diagnosis. On the contrary – Respondent may offer evidence, or point to existing evidence in the record, that contradicts or weakens a petitioner's evidence, and in so doing demonstrate that the petitioner cannot meet her overall burden. *Stone v. Sec'y of Health & Human Servs.,* 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[o]ur decisions support the commonsense proposition that evidence of other possible sources of injury can be relevant not only to the "factors unrelated" defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question"); *La Londe v. Sec'y of Health & Human Servs.,* 110 Fed. Cl. 184, 198 (2013) ("[r]egardless of whether the burden ever shifts to the respondent, the special master may consider the evidence presented by the respondent" when determining if petitioner's initial burden has been met), *aff'd*, 736 F.3d 1334 (Fed. Cir. 2014). In this case, Respondent's challenge to the proper diagnosis of Petitioner's illness does not amount to an attempt to establish a causal "factor unrelated," and therefore in considering such issues I do not find that Respondent is burdened with proving them.

VI. ANALYSIS

A. *Dispute Over Petitioner's Diagnosis*

Both sides devoted a substantial portion of the hearing to addressing whether the HPV vaccine could be shown to cause GBS (or more precisely the AIDP variant of GBS). The medical records in this case, however, suggest a more immediate question: whether Ms. Blackburn had GBS at all, and/or whether her illness began only *after* her receipt of vaccination. Ms. Blackburn and her counsel did not originally plead a claim based upon significant aggravation of a pre-existing condition, and have otherwise repeatedly denied that they do so now. *See generally Hirmiz v. Sec'y of Health & Human Servs*., No. 06-371V, 2014 WL 7204716, at *9-10 (Fed. Cl. Dec. 4, 2014) (failure to amend petition before trial to add significant aggravation was fatal to claim, despite fact that evidence offered at trial incidentally supported such a claim). Therefore, if the illness Petitioner suffered from was different from what was alleged, began before she ever received the HPV vaccination, and/or was not itself triggered or aggravated by the vaccination – then Petitioner's entire claim cannot succeed.[49]

[49] In his testimony, Dr. Steinman did claim that his theory for how the HPV vaccine could have caused Ms. Blackburn's illness still applied even if she suffered from CIDP rather than the AIDP variant of GBS. Tr. at 186-89. However, he admitted that his theory was heavily focused on AIDP being the correct diagnosis, and he did not testify that Ms. Blackburn's pre-vaccination symptoms were aggravated by the Gardasil vaccine – nor did he ever

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the precise nature of a petitioner's injury before engaging in the *Althen* analysis. *Broekelschen*, 618 F.3d at 1346. Indeed, the fact that a claimed injury predates the vaccination can defeat a Vaccine Program claim entirely. *Shalala v. Whitecotton*, 514 U.S. 268, 274-75(1995) (Vaccine Act claimant who demonstrates she experienced symptoms of injury after receipt of vaccination does not succeed in her claim if the evidence fails to indicate that she had no symptoms of injury before her vaccination); *Locane v. Sec'y of Health & Human Servs.*, 99 Fed. Cl. 715 (2011) (petitioner's Crohn's disease began prior to her vaccinations and therefore vaccine causation could not be established). Since "each prong of the *Althen* test is decided relative to the injury" (*Broekelschen*, 618 F.3d at 1346), determining facts relating to the claimed injury can be significant in a case like this, where the petitioner has symptoms predating her vaccination that could be consistent with an illness other than that alleged as the injury. Indeed, as another special master recently noted, "where the respondent presents evidence of an alternative diagnosis, the special master may consider the respondent's evidence of that alternative diagnosis as part of the master's evaluation of the petitioner's *prima facie* showing of an injury, potentially mooting the *Althen* causation test." *Hirmiz,* 2014 WL 7204716, at *14.

Thus, before determining whether Ms. Blackburn has met each of the individual *Althen* prongs, I address whether she has established by preponderant evidence that she suffered from the AIDP variant of GBS, and whether her illness began prior to her vaccination.

1. GBS/AIDP vs. CIDP – As noted in the Koningsveld article[50] included in the medical literature offered by Petitioner, GBS is an acute variant of an inflammatory demyelinating polyneuropathy. Pet'r's Ex 36 at 1. It is a rapidly progressing and ascending motor neuron paralysis frequently seen after infection[51] (*Dorland's* at 1832), and it is typically monophasic. Pet'r's Ex. 37 at 1. From a clinical diagnostic standpoint, an individual with GBS reaches the peak severity of his symptoms (nadir) within four weeks from initial onset. Koningsveld at 138.

---

maintain that her earlier symptoms had anything to do with her post-vaccination illness. More significantly (and in keeping with the fact that Petitioner does not advance a significant aggravation claim in this case), Petitioner did not offer any testimony or medical literature explaining how (assuming Ms. Blackburn's illness predated vaccination) the HPV vaccine could have worsened a pre-existing condition. I therefore could not find that Petitioner has offered preponderant evidence to satisfy a significant aggravation off-Table claim, even if such a claim had been alleged.

[50] Ruts, L., Van Koningsveld, R., & Van Doorn, P.A., *Distinguishing Acute-Onset CIDP from Guillain–Barré Syndrome with Treatment Related Fluctuations*, 65 Neurol. 1, 138-40 (2005) (Pet'r's Ex. 36) [hereinafter "Koningsveld"].

[51] Odaka, M., Yuki, Nobuhiro, Y., Hirata, K., *Patients with Chronic Inflammatory Demyelinating Polyneuropathy Initially Diagnosed as Guillain–Barré Syndrome*, 250 J. Neurol. 913-16(2003) (Pet'r's Ex. 35) [hereinafter "Odaka"].

AIDP is a common GBS variant (and is sometimes thought of as synonymous with the disease). P. Dyck & P.K. Thomas, 2 *Peripheral Neuropathy* 2199 (4th ed. 2005) [hereinafter "Dyck & Thomas"]. It shares the acute and monophasic characteristics of other GBS variants. Ted M. Burns, M.D., *Guillain-Barré Syndrome*, 28 Seminars in Neurol. 152-67 (2008) (Resp't's Ex. G at 3) [hereinafter "Burns"]; Dyck & Thomas at 2222. AIDP is considered to be immune-mediated, meaning that a triggering event (which may or may not be identified) occurs, leading to an aberrant immune response which in turn causes a breakdown of the blood-nerve barrier and destruction of the myelin sheath (and secondarily also the axon) resulting in the clinical presentation seen in an individual with AIDP. Wilson, H. J., *The Immunobiology of Guillain–Barré Syndromes*, 10 J. Peripheral Nervous System 94-112 (2005) (Resp't's Ex. O). Some of the symptoms included in the clinical definition of AIDP are: (a) numbness and tingling in the feet progressing in an ascending fashion to the arms; (b) progressive and ascending weakness; (c) areflexia; and (d) cranial nerve dysfunction/facial weakness. Amato, A. & Russell, J., 227 *Neuromuscular Disorders* 213-14 (1st ed. 2006) (Pet'r's Ex. 33) [hereinafter "Amato"]; Van Doorn, P.A., Ruts, L., Jacobs, B.C., *Clinical Features, Pathogenesis, and Treatment of Guillain–Barré Syndrome*, 7 Lancet Neurol. 939 (2008) (Resp't's Ex. F) [hereinafter "Van Doorn"]. Tests that can confirm or lend support to the diagnosis include a lumbar puncture (to determine elevation of protein levels in the cerebrospinal fluid) and EMG/nerve conduction studies. Van Doorn at 940, 950.

CIDP is also a demyelinating condition with symptoms similar to AIDP, but typically presents in either a chronically progressive or relapsing-remitting form. Dyck & Thomas at 2221-22. A CIDP patient's neurologic symptoms develop over weeks and months (with eight weeks viewed as the minimum clinical period for a CIDP diagnosis), and an antecedent infection is much less commonly identified as the initiating factor for the illness. *Id.* Facial muscle weakness and autonomic nervous system impairment are also less common in CIDP than in GBS. *Id*. at 2222-23.

Despite their differences, distinguishing between the two diseases is difficult – in particular during their early phases. Koningsveld at 138; Odaka, M., Yuki, N., Hirata, K., *Patients with Chronic Inflammatory Demyelinating Polyneuropathy Initially Diagnosed as Guillain–Barré Syndrome*, 250 J. Neurol 913-16 (2003) (Pet'r's Ex. 35) [hereinafter "Odaka"] ("the difference between GBS and CIDP in some patients may be blurred during the first [four] weeks" – "physicians could classify patients with CIDP within [four] weeks of onset as GBS"). Patients whose symptoms are initially believed to be compatible with GBS may later be diagnosed with CIDP based on the subsequent course of illness. Koningsveld at 138. And when treatment is administered early in the course of disease, patients may actually experience a simulated GBS episode within the overall context of CIDP (thus causing further diagnostic confusion). Odaka at 914.

It is important for clinicians to attempt to distinguish between CIDP and GBS as early in

the course of illness as possible because of differences in treatment and prognoses. Koningsveld at 138, 140. Although IVIG treatments are utilized successfully in resolving both GBS/AIDP and CIDP, corticosteroid treatments "provide clear-cut benefit for most patients with CIDP, whereas they were shown to be ineffective in AIDP." Dyck & Thomas at 2223; *see also* Jean-Michel Vallat et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Diagnostic and Theraputic Challenges for a Treatable Condition,* 9 Lancet Neurol. 403-12 (2010) (Res'p't Ex. I at 7-8)**;** Mori, K. et al., *Chronic Inflammatory Demyelinating Polyneuropathy Presenting with Features of GBS*, 58 Neurol. 979 (2002) (Pet'r's Ex. 34) (corticosteroid therapy is not considered beneficial for GBS)**;** Odaka at 916; Van Doorn at 939-50.

Recurrences or treatment-related fluctuations have been seen in individuals with GBS. Hadden, R.D.M., *Deterioration After Guillain–Barré Syndrome: Recurrence, Treatment-Related Fluctuation, or CIDP?*, 80 Neurol. Neurosurg. Psychiatry 3 (2009) (Pet'r's Ex. 37) [hereinafter "Hadden"]. A treatment-related fluctuation has been defined "as significant deterioration within [two] months after disease onset, following post-treatment improvement or stabilization," which is thought to occur as a result of "relatively prolonged autoimmune activation outlasting the effect of treatment." *Id.* Recurrent GBS is distinguished from CIDP by "very long asymptomatic periods with return of tendon reflexes, more frequent antecedent illness, rapid onset, frequent facial weakness and normal CSF protein within [one] week of onset." Hadden at 3; Odaka at 916.

Both experts generally agreed on the above, but had slightly different interpretations of various elements of the two diseases. Dr. Steinman expressed the view that GBS and CIDP are on the same spectrum – with AIDP simply constituting a more acute form of demyelinating polyneuropathy than CIDP. Tr. at 61-62, 64; *see also* Marinos C. Dalakas, *Advances in the Diagnosis, Pathogenesis and Treatment of CIDP*, 7 Nature Reviews Neurol. 507-17 (2011) (Resp't's Ex. J) [hereinafter "Dalakas"]. He thus suggested that distinguishing between the two based on onset and/or length of progression of symptoms reflected an "arbitrary separation from subacute and chronic demyelinating polyradiculoneuropathy." Tr. at 65 (quoting Richard A.C. Hughes & Jeremy H. Rees, *Clinical and Epidemiologic Features of Guillain-Barré Syndrome*, 176 (Supp. 2) J. Infectious Diseases S92 (1997) (Resp't's Ex. L at 2)).[52] Nevertheless, Dr. Steinman acknowledged that "CIDP differs from GBS [] by its time course, mode of evolution, prognosis, and responsiveness to steroids." Tr. at 64.

Dr. Steinman asserted that the presenting symptoms for each disease were different. Thus, areflexia and symmetry are classic aspects for the presentation of an individual with CIDP,

[52] Dr. Steinman elaborated on this point by suggesting that neurologists are famous for wanting to either "lump or split" (*see* Tr. at 62), and thus temporal distinctions between the progressions of both conditions reflected a desire by neurologists responsible for defining clinical facets of each to "draw the line" rather than meaningfully distinguish what are otherwise closely-related illnesses. *Id*. at 65.

but not necessarily present in a patient with GBS. Tr. at 66, 68. He further testified that in his experience it typically takes areflexia approximately one month to six weeks to develop in an individual with CIDP, after which reflexes would not return. *Id*. at 66. As a result, Dr. Steinman indicated that he would be reluctant to diagnose a patient that had reflexes with CIDP – and he would view a patient whose reflexes recovered, but whose other related symptoms had not dissipated and appeared chronic, as still recovering from AIDP, rather than as manifesting CIDP. *Id*. at 63-64.

Dr. Chaudhry, by contrast, emphasized that in his view (which he testified was consistent with the view of "all neuromuscular physicians"), CIDP and GBS represent two separate (if related) conditions rather than opposing points on the same disease spectrum – and that as such it would be incorrect to view CIDP simply as a chronic form of GBS. Tr. at 208-09. Rather, they are distinguishable in terms of their presentation, treatment, and prognosis. *Id.* at 208. Thus, GBS (and its most common variant in the Western world, AIDP) is characterized by its rapid and acute progression, with subsequent slow recovery over a period of time – there is typically no waxing and waning course.[53] *Id.* at 202-04. He also noted that IVIG is currently the standard treatment for GBS. *Id.* at 204-05.

CIDP patients also experience weakness and numbness, exhibit loss of reflexes, exhibit high spinal fluid proteins, and demyelination under conduction. Tr. at 206. A distinguishing characteristic of CIDP, however, is its chronic nature, defined by lengthy relapsing and remitting phases.[54] *Id*. Dr. Chaudhry expressed the view that the clinical diagnostic definition of CIDP as needing to last at least eight weeks was misleading – in his experience the course of illness is typically far longer. *Id.* at 206-07. Indeed, Dr. Chaudhry testified that he has observed patients with CIDP see their progression of symptoms take more than a year to get to the point where treatment was considered or warranted. *Id.* Dr. Chaudhry also stressed the significance of prednisone as highly indicated for the successful treatment of CIDP. *See generally Id.* at 205-07.[55] According to Dr. Chaudhry, Prednisone is not used to treat GBS – and in Dr. Chaudhry's opinion it would be malpractice to administer it to a GBS patient. *Id.* at 236.

---

[53] Dr. Chaudhry acknowledged that there is a subcategory of GBS referred to as "relapsing Guillain-Barré," but he testified that in his view this diagnostic classification is limited to those patients who have an attack of GBS, recover completely, and then have another attack months or years later. Tr. at 257.

[54] In fact, Dr. Chaudhry stated, CIDP typically lasts throughout an individual's lifetime, with most patients requiring ongoing treatment, although a few patients do go into remission (approximately one-third probably less). Tr. at 207.

[55] A distinct diagnostic criterion for CIDP (at least in 1975 when it was first described by Dr. Peter Dyck), before the availability of physiology and nerve conduction studies, was steroid responsiveness to a patient's symptoms. Tr. ay 205-06. Dr. Chaudhry noted that CIDP was actually termed "steroid responsive chronic relapsing remitting polyneuropathy" in the landmark 1975 paper in the journal *Neurology*. *Id*. at 206. Other medications used for treatment of CIDP (CellCept, Rituximab, Imuran, and Cyclosporine), Dr. Chaudhry testified, do not have evidence-based medicine supporting their effectiveness, but are often employed because they can have a steroid-sparing effect (allowing for a reduction in the amount of steroid that is required to be taken by the patient). *Id.* at 207.

2. Petitioner's Medical History Suggests Her Actual Illness was CIDP – Although there is contradictory evidence in the medical records, those record by themselves (viewed in their totality and without the benefit of expert interpretation) suggest that Ms. Blackburn more likely than not suffered from CIDP, rather than the AIDP variant of GBS.

Ignoring for the moment Ms. Blackburn's pre-vaccination medical history,[56] the records establish that Ms. Blackburn began experiencing symptoms no earlier than August of 2009. Pet'r's Ex. 2 at 9-11; Pet'r's Ex. 8 at 4-5. Even after intense treatment in August and September of 2009, however – about eight weeks after Ms. Blackburn's HPV vaccination (a period almost twice as long as the four-week onset of GBS symptoms in most cases (*see, e.g.*, Koningsveld at 138)) – it became evident that Ms. Blackburn was again ill. Pet'r's Ex. 7 at 14-15; Pet'r's Ex. 5 at 363.

Her symptoms continued to wax and wane well into the spring of 2010, nine months later. Pet'r's Ex. 7 at 6-7. Indeed, the very fact that Petitioner continued to experience symptoms *despite* the initial AIDP diagnosis and immediate treatment specific to that illness was a factor that lead Dr. Bromberg to conclude that she likely was suffering from CIDP. *Id.* at 2, 8-9. Because AIDP and CIDP can be confused in their early stages, it is not surprising that the treating physicians who first examined Ms. Blackburn between August and October of 2009 reached different conclusions from those treating her six months later.

The successful alteration of Ms. Blackburn's treatments also strongly supports the CIDP diagnosis. By April 2010, the fact that Ms. Blackburn's symptoms had still not fully cleared prompted Dr. Bromberg to try a prednisone course, based on the reasonable medical inference that a demyelinating condition that waxed, waned, and/or relapsed over several months was not sufficiently acute or monophasic to constitute a GBS variant. Pet'r's Ex. 7 at 8. The medical literature strongly associates prednisone with the successful treatment of CIDP. *See, e.g.*, Dalakas at 508. And in fact the prednisone worked. Pet'r's Ex. 7 at 2-4.

Petitioner unconvincingly objects to the CIDP diagnosis that the medical history reflects. She argues that I should give great weight to the views of some treating physicians (in particular, those who treated Ms. Blackburn in the two to three months after her vaccination and/or made

---

[56] Ms. Blackburn's pre-vaccination history lends some support to the CIDP diagnosis. She first reported foot and hand numbness in October 2008 (*see* Pet'r's Ex. 4 at 2-3), and complained of numbness and tingling on the actual vaccination date in July 2009 (*see* Pet'r's' Ex. 2 at 11-12) – suggesting her condition existed for a long time prior to its significant flare-up in August 2009 (*see Id.* at 9-10). Admittedly, however, Ms. Blackburn's pre-vaccination treatment history also contains evidence less supportive of CIDP, as certain clinically-significant diagnostic clues (in particular, areflexia) did not point toward any severe neuropathy (*see, e.g.*, Pet'r's Ex. 8 at 18), and her post-vaccination symptoms were self-evidently more severe than what she had experienced in the months before. Pet'r's Ex. 2 at 9. I therefore do not find that the pre-vaccination medical history (taken alone, without the assistance of an expert's review) is particularly strong evidence for either side's position – although, when expert interpretation of that record is added to my analysis, the pre-vaccination history becomes far more significant.

the initial AIDP diagnosis) but not others. Tr. at 118-19, 126-28. Petitioner particularly maintains that the determinations of Dr. Whitesell (which the record unequivocally establishes were made within a month or two of Ms. Blackburn's vaccination) should predominate over a determination made by Dr. Bromberg – despite the fact that Dr. Bromberg's evaluations and observations took into account what had happened during the months that had passed from the time Ms. Blackburn first complained of post-vaccination symptoms.

Unquestionably the views of treating physicians are important, but they are also properly subject to evidentiary weighing. *Capizzano*, 440 F.3d at 1326. The overall course of Ms. Blackburn's treatment history supports giving less weight to the determinations of the treating physicians who first saw her. Dr. Whitesell and others reached immediate conclusions about the nature of Ms. Blackburn's illness without the benefit of the evidence Dr. Bromberg later relied on, including the results of trying a different medication used almost exclusively for CIDP. Significantly, there is nothing in the record suggesting that any other treating physicians who saw Ms. Blackburn after the prednisone's efficacy was established in treating her symptoms disagreed with Dr. Bromberg's conclusion regarding her diagnosis. Dr. Whitesell's initial diagnosis of AIDP may, at the time, have been reasonable and supportable based on the immediate evidence at hand – but ultimately it proved incorrect.[57]

All in all, I must consider the treatment record as a whole in evaluating the evidence offered to establish Petitioner's illness. § 13(a)(1). Here, the record alone supports the conclusion that the corrected diagnosis – CIDP – was more accurate, given (a) the relapsing course of Ms. Blackburn's symptoms, and (b) the effectiveness of the prednisone treatment.

3. Respondent's Expert Persuasively Confirmed Both the CIDP Diagnosis and its Pre-Vaccination Onset – In a case such as this, where expert testimony is offered to interpret medical records and/or contemporaneous tests performed on the petitioner, a special master necessarily determines the persuasiveness of each competing expert. *See, e.g.*, *Carrino v. Sec'y of Health & Human Servs.,* No. 08-0266-V, 2013 WL 3328903, at *11-20 (Fed. Cl. Spec. Mstr. June 6, 2013) (finding Respondent's expert's review of medical history and specific test results more persuasive than Petitioner's expert's review, in connection with determination that Petitioner did not suffer from GBS). This flows naturally from a special master's duty to evaluate expert credibility in the process of weighing the evidence. *Porter v. Sec'y of Health & Human Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("[t]he Federal Circuit has "unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

[57] I also note that Dr. Whitesell's opinion was itself somewhat qualified, conclusory, and not corroborated with any explanatory basis. *See, e.g.*, Pet'r's Ex. 3 at 66 ("I feel that this likely still represents slowly improving Guillain-Barré which can sometimes have a waxing and waning course . . . I feel that an alternative diagnosis of CIDP is less likely").

Both experts were qualified to testify to the matters in dispute generally, but Dr. Chaudhry was better qualified than Dr. Steinman to opine on the nature and treatment of GBS and CIDP, and his testimony was more credible. His demonstrated, day-to-day experience in seeing patients and reviewing the results of tests most relevant to diagnosing demyelinating diseases rendered his opinion particularly trustworthy. He also persuasively grappled with the record, acknowledging contrary evidence rather than dismissing it out of hand. Dr. Steinman, by contrast, has had far less direct experience with GBS or CIDP patients over the past several years, and less frequently reviews the results of (let alone performs) EMGs and nerve conduction studies. His core competency is with MS and central nervous system diseases. While such illnesses have some relationship to the matters in dispute (as they also involve demyelinating conditions with some common symptoms) they are not equivalent. Petitioner called upon Dr. Steinman in this case to wear two hats as an expert – that of a diagnostic expert as well as a theory/*Althen* prong one expert – but the latter fit him far better than the former, and I found him less persuasive on diagnostic topics.

Dr. Steinman's reduced expertise on matters pertaining to the diagnosis and treatment of peripheral neuropathies was evident during the hearing (and particularly when he attempted to minimize the impact of evidence harmful to the Petitioner's case). For example, Dr. Steinman strained in arguing that there was nothing notable about the positive results of Ms. Blackburn's prednisone treatments, asserting that corticosteroids were simply another option for any GBS-related illness, and would invariably make a patient feel better regardless of their clinical impact on the underlying disease. Tr. at 73. But this is contrary to the medical literature submitted by both parties, all of which strongly suggests that prednisone is indicated only for the treatment of CIDP. Indeed, Dr. Chaudhry (who plainly possesses substantially more experience in diagnosing and treating both GBS and CIDP) characterized the use of prednisone for treatment GBS patients as medical malpractice.

Dr. Steinman was similarly unpersuasive in his effort to frame CIDP as little more than a severe, lasting case of AIDP. Tr. at 62. While it is true that the two diseases have much in common and (especially early in their course) can be easily confused, they are distinguishable – and, given the difference in treatment and prognosis, *need* to be distinguished in order to better assist patients in recovering from either. *See, e.g.*, Koningsveld at 138; Odaka at 913, 916. But Dr. Steinman did not establish that the recurrence and persistence of Ms. Blackburn's symptoms, after her seemingly successful treatment in August through September of 2009, was properly attributed to either treatment fluctuations or a "recurring" form of AIDP. The medical record in fact does not suggest that any treatment related fluctuations occurred.[58] And beyond some

[58] Notations in Ms. Blackburn's medical records indicate that she received "2 g/kg of IVIG treatment over three days" (Pet'r's Ex. 7 at 7) and there is also a notation taken from pharmacy records stating that she "received 35 g[rams] of IVIG on 08/25/2009, 35 g[rams] of IVIG on 08/26/2009, and 30 mg of IVIG on 08/27/2009." *Id.* at 25. Dr. Chaudhry noted that there appeared to be a typographical error in the pharmacy notation – Ms. Blackburn would have more likely received 30 grams (not milligrams) for her final IVIG dose, adding up to a total dosage of 100 grams. Tr. at 224. In Dr. Chaudhry's view, this is consistent with the standard dose for someone of her stature (as

medical literature stating that a fluctuating form of AIDP exists, the only support for this alternative diagnosis is Dr. Steinman's *ipse dixit*, rather than evidence drawn from the record.

Dr. Steinman failed to provide an overarching explanation for Ms. Blackburn's illness based on the totality of her medical history. Thus, he placed excessive reliance on Dr. Whitesell's October 2009 determination that CIDP was not likely the correct diagnosis for Petitioner – while giving less weight to the subsequent determination of Dr. Bromberg (as well as every other treating physician thereafter) that CIDP was in fact the correct diagnosis. Dr. Whitesell's opinion itself contains no record support or corroboration explaining why she discounted the possibility of a CIDP diagnosis. *See* Pet'r's Ex. 3 at 66. Dr. Bromberg, by contrast, had the benefit of months more of evidence regarding Petitioner's condition, plus the fact that the test he proposed for determining if CIDP were the correct diagnosis (the prednisone treatment) worked. Dr. Steinman generally found more significant the initial diagnosis Ms. Blackburn received – a diagnosis that circumstances, and time, reasonably led her subsequent treating providers to re-evaluate. *See, e.g.*, Pet'r's Ex. 7 at 8. Dr. Steinman's interpretation favored the position that once AIDP had been diagnosed, it remained the "correct" diagnosis even if later treatment evidence contradicted it. Tr. at 63.

Dr. Steinman was also selective in what symptoms he deemed significant versus those he ignored. Thus, he overemphasized areflexia as a symptom of CIDP (thus rendering its absence from Ms. Blackburn's pre-vaccination medical history a telling fact).[59] Yet the literature offered by the parties more definitively identifies areflexia as a presenting symptom of GBS. *See, e.g.,* Burns at 3 (in GBS, "[w]idespread areflexia or hypoflexia is the rule"); Amato at 214 (Pet'r's Ex. 33 at 2). In so doing, Dr. Steinman ignored the fact that Ms. Blackburn did not display upper limb areflexia as of mid-August 2009 (*see* Pet'r's Ex. 4 at 4-5), even though she would then have reached, if not be approaching, the nadir of her GBS (assuming, as Dr. Steinman opined, that her illness began with her July vaccination). At the same time, Dr. Steinman consistently downplayed the importance of cranial nerve weakness or dysfunction as a presenting symptom of GBS (Tr. at 71) – even though it is considered such (Van Doorn at 939-50) – and thus sidestepped the fact that Ms. Blackburn never displayed this particular symptom during her purportedly acute AIDP phase in August and September of 2009. Tr. at 91.

---

the medical records indicate that she was five foot four inches and weighed approximately 104 pounds around this period of time. *Id.*; *see also* Pet'r's Ex. 7 at 12. The medical literature corroborates Dr. Chaudhry's view. *See, e.g.*, Dyck & Thomas at 642-43. Nothing else in the record indicates that Ms. Blackburn did not receive a full course of IVIG.

[59] In any event, as Dr. Chaudhry later explained, individuals suffering from CIDP often experience a slow, long-term ramping up of the illness, during which common presenting symptoms might remain mild for a long period of time, and therefore the absence of clear areflexia before vaccination does not discredit the ultimate conclusion (bulwarked by a comprehensive review of the record) that CIDP was the correct diagnosis. Tr. at 206-7.

Dr. Chaudhry, by contrast, rooted his opinion in a complete view of the record. Having treated numerous CIDP patients (Tr. at 196-97), he was well qualified to observe that a patient may experience a number of mild neuropathic symptoms long before they are actually diagnosed with CIDP (and even before such a diagnosis might be proper). *Id*. at 206-07, 226. Because of the chronic nature of their symptoms, CIDP patients can "live" with the condition without hospitalization for a long time, as opposed to the acute, fast-moving nature of AIDP. Dr. Chaudhry credibly explained why Ms. Blackburn's early examinations and test results did not display all of the formal clinical indicia of CIDP. *Id*. at 212-13.[60] There were enough other signs to see, in retrospect, a relationship between Ms. Blackburn's pre-vaccination neuropathic symptoms and her illness's subsequent course. *Id*. at 227, 249-50.

Dr. Chaudhry was also persuasive in identifying inconsistencies in Ms. Blackburn's history from August to September 2009 that undercut the initial AIDP diagnosis. Through his reading of contemporaneous EMG test results, he pointed out convincingly that early symptoms and test results actually better supported the CIDP diagnosis – the nerve damage, for example, evident from Ms. Blackburn's August EMG (*see* Pet'r's Ex. 4 at 7) was too extensive to have occurred in the weeks immediately after her July 23rd vaccination, but was more likely evidence of a pre-existing illness that had to have begun before that date. Tr. at 216, 218.

At bottom, Dr. Chaudhry was more persuasive in commenting on what was (or was not) relevant in diagnosing GBS and CIDP – based on evidence from contemporaneous medical records before and after Petitioner's vaccination. In so doing, he convincingly offered an interpretation of the medical history that Petitioner has not rebutted – that it is improbable that Petitioner suffered from AIDP only beginning with her Gardasil vaccination.

B. *Application of Althen Prongs*

Although my determination that Ms. Blackburn did not suffer from AIDP, and that her illness began before her vaccination, potentially obviates the need for an extensive *Althen* analysis, I nevertheless consider below each of its prongs based on the evidence presented.

1. Prong One: Can the HPV Vaccine Cause GBS and/or CIDP? Petitioner offered little direct evidence supporting her theory that HPV vaccine can cause GBS, other than VAERS reports,[61] or research and case studies involving other vaccines which Petitioner argues

[60] Respondent's medical literature supported the idea that areflexia is not necessarily a *sine qua non* symptom of CIDP even if it is associated with the disease. *See, e.g.*, Dalakas at 2-3 (CIDP characterized generally by "a progressive, symmetric, proximal and distal muscle weakness, paresthesias, sensory dysfunction, and impaired balance").

[61] "VAERS" refers to the Vaccine Adverse Event Reporting System. The value of a specific VAERS report as evidence in proving causation in a Vaccine Program case is extremely limited. As another special master has observed,

are analogous.[62] There is, however, persuasive scientific evidence on the other side of the question. As Dr. Steinman admitted, the Institute of Medicine ("IOM") has determined that "[t]he epidemiologic evidence is insufficient or absent to assess an association between HPV vaccine and GBS." Tr. at 81-82; *see also* Adverse Effects of Vaccines at 512.[63] Nevertheless, there is no specific category of evidence that a petitioner must offer to establish a medical theory supportive of a causation finding (*Althen*, 418 F.3d at 1280), and therefore Petitioner's inability to marshal this kind of specific scientific proof does not mean she cannot meet the requirements of the first *Althen* prong.

Molecular mimicry theories have been accepted in other Vaccine Program cases as a general framework for explaining the development of certain autoimmune diseases (although such acceptance has not always resulted in entitlement decisions favorable to petitioners). *Tompkins v. Sec'y of Health & Human Servs.*, No. 10-261V, 2013 WL 3498652, at *22 (Fed. Cl. Spec. Mstr. June 21, 2013) ("[t]he molecular mimicry theory is the one most widely accepted for the agents most frequently accepted as causal"), *motion for review den'd, Tompkins v. United States,* 117 Fed. Cl. 713 (2014). Dr. Whitton conceded that molecular mimicry may explain vaccine-induced demyelinating injuries, as long as its basic requirements are met (Tr. at 362), and Respondent ultimately did not seriously question that this theory offers a potential explanation for how autoimmune diseases like GBS develop. *See* Resp't Mem. at 23-27. But accepting that molecular mimicry has previously been found to be a reasonable general theory[64]

> VAERS is a stocked pond. It only contains reports (many of which are unverified or incomplete) of adverse events after vaccinations. VAERS contains no reports or data about the relative rate of these same events in individuals who have not been vaccinated. Thus, the number of specific adverse events, such as GBS, reported after any vaccine, is meaningless without information about the background rate of that adverse event and information about the number of vaccines administered.

Tompkins v. Sec'y of Health and Human Servs., No. 10-261V, 2013 WL 3498652, at *16 (Fed. Cl. Spec. Mstr. June 21, 2013), *motion for review den'd, Tompkins v. United States,* 117 Fed. Cl. 713 (2014).

[62] Respondent also questioned Petitioner's reliance on research pertaining to central nervous system diseases, such as MS in humans and EAE in rodents. Resp't's Pre-Hr'g Mem. (ECF No. 62) at n. 7. Respondent further questioned the evidence showing homology between Y and F in mouse models, arguing that it did not constitute scientifically reliable evidence that they are interchangeable when dealing with human models. Respondent's Reply to Pet'r's Post-Hr'g Brief (ECF No. 79) at 5. While acknowledging that animal models have been found to be sufficient in some Vaccine Program cases, I do not specifically address this contention as it does not have a direct bearing on my decision in this case.

[63] IOM evidence is especially reliable and useful in gauging whether a vaccine "can cause" a given injury. *See generally Crutchfield v. Sec'y of Health & Human Servs.,* No. 09-0039V, 2014 WL 1665227, at *16 (Fed. Cl. Spec. Mstr. Apr. 7, 2014) ("[d]uring the 25–year history of the Vaccine Act, special masters have consistently relied upon the reports of the Institute of Medicine, and reviewing judges have consistently indicated approval of such reliance").

[64] I do not mean to say that the molecular mimicry theory is in all cases reasonable. On the contrary – the theory has at times been misused by certain experts, who have invoked it as a universal mechanism that can explain virtually any demyelinating or other autoimmune condition. *See, e.g.*, *Hennessey v. Sec'y of Health & Human Servs.,* 91 Fed.

does not complete my analysis under *Althen* prong one – the theory offered must have application to *this case*. *See Caves*, 100 Fed. Cl. at 135 (noting that the *Althen* requirement to offer medical theory would be meaningless if petitioner did not need to apply molecular mimicry specifically to her case); *Broekelschen*, 618 F.3d at 1345.

Respondent generally questioned whether Dr. Steinman's corrected-on-the-witness-stand homology theory (which posits a mechanism for how the HPV vaccine could result in a peripheral neuropathy) has been shown to be scientifically reliable. Resp't Pre-Hr'g Mem. (ECF No. 62) at 18; Tr. at 168-69. A petitioner need not necessarily demonstrate the precise homology involved when invoking molecular mimicry as the mechanism for explaining how a particular vaccine could cause injury. *See, e.g.*, *Salmins v. Sec'y of Health & Human Servs.*, No. 11-140, 2014 WL 1569478 (Fed. Cl. Spec. Mstr. Mar. 31, 2014) (granting entitlement in HPV/GBS case when petitioner relied on molecular mimicry without showing homology). But because the homology question was hotly disputed, I will review the evidence on the point pro and con.

Dr. Steinman's homology theory is inconsistent – particularly the manner in which he conflates autoimmune responses involving antibodies produced by B cells with those involving T cells. *See* Tr. at 17, 46; Pedro J. Ruiz et al., *Microbial Epitopes Act as Altered Peptide Ligands to Prevent Experimental Autoimmune Encephalomyelitis*, 189 J. Experimental Med. 1275 (1999) (Pet'r's Ex. 21). The medical literature cited by the parties does not support the notion that this homology "works" regardless of whether the autoimmune response involves T cells or B cell antibodies.[65] If limited to the context of T cell mimicry, however, there is better support for his F and Y equivalence argument in the context of an attack on MBP.[66] Tr. at 157-60.

Cl. 126, 134-35 (2010) (noting expert's overly broad application of the molecular mimicry theory made it meaningless).

[65] In his expert reports and testimony, Dr. Steinman invoked Wucherpfennig for the proposition that the FKN sequence is of particular importance to the homology between T cells and B cell antibodies, on the one hand, and MBP on the other. Tr. at 44. He then immediately jumped to a different study (Grada M. van Bleek & Stanley G. Nathanson, *The Structure of the Antigen-Binding Groove of Major Histocompatibility Complex Class I Molecules Determines Specific Selection of Self-Peptides*, 88 Immunology 11032 (1991) (Pet'r's Ex. 32) [hereinafter "van Bleek"]) to support his assertion that F and Y are "pretty much interchangeable." Tr. at 46-47. The van Bleek study, however, demonstrates only that Y and F are interchangeable in the specific context of binding T cells to mouse MHC class I molecules. Van Bleek at 11035.

Dr. Whitton challenged the relevance of this study to humans (Tr. at 305-06), referencing Wucherpfennig (Pet'r's Ex. 18 at 1117-18) to demonstrate that substituting Y for F has a "profound effect on immune recognition" in humans. Tr. at 311. The Wucherpfennig study specifically finds that F and Y are *not* interchangeable in the context of human autoantibody recognition. Wucherpfennig at 1116. Indeed, Wucherpfennig suggests that a YKN sequence is too limited to trigger cross-reactive antibodies (even assuming arguendo that F and Y were interchangeable), because "autoantibody recognition of microbial peptides required sequence identity over a stretch of four or five amino acids." *Id.* at 1119.

[66] The HFFK sequence of MBP is its "core motif" recognized within the T cell epitope. Pet'r's Ex. 21 at 1275. Within this sequence, "K91 is the major TCR contact site, while F90 is a major anchor to MHC." *Id*. Thus, the cited medical literature supports the concept that the two-acid FK sequence is indeed critical, and that limited peptide

Respondent argued that the FK/FY sequence is too common. Tr. at 313-14. If a two-protein homology is sufficient for cross-reactivity, she asserted, then the majority of microbes could serve as the basis for molecular mimicry in so many circumstances that the theory would be "so broad as to be meaningless." *Hennessey*, 91 Fed. Cl. at 135; *Tompkins*, 2013 WL 3498652, at *23. Petitioner responded that because *structural* similarity is what drives Dr. Steinman's molecular mimicry theory, little *sequential* homology is required for T cell mimicry to occur. *See* Rafael L. Ufret-Vincenty, *In Vivo Survival of Viral Antigen-Specific T Cells that Induce Experimental Autoimmune Encephalomyelitis*, 188 J. Exp. Med. 1726 (1998) (Pet'r's Ex. 23).

Respondent also challenged the precise mechanism offered by Petitioner to explain how the autoimmune process resulting in GBS would function. Dr. Steinman's homology analysis depends upon MBP as the target antigen. *See, e.g.,* Pet'r's Pre-hr'g Mem. (ECF No. 60) at 15-16**;** Tr. at 162-63. But as Dr. Whitton testified, available scientific evidence (reflected in the literature offered in this case) suggests that MBP is *not* the target antigen in autoimmune responses that result in a peripheral neuropathy like GBS. Tr. at 317, 327. Rather, other protein structures on the peripheral myelin have been demonstrated as far more likely targets of a demyelinating process. *Id.* at 290-94. Indeed, the "most solid evidence for a molecular mimicry" mechanism producing GBS (as Dr. Steinman acknowledged) involves an immune response against gangliosides. *Id.* at 162-63 (Dr. Steinman admitting that studies of the "*Campylobacter* story" involve attacks against gangliosides, "a different molecular mimic" from MBP); Komagamine at 394; *see also Daily v. Sec'y of Health & Human Servs.*, No. 07-173V, 2011 WL 2174535, at *3 (Fed. Cl. Spec. Mstr. May 11, 2011) ("[a] large body of literature links gangliosides and antibodies against certain gangliosides as causal agents of GBS").

In support of her contention that MBP is the target antigen, Petitioner offered only the Cornblath study, which found increased MBP in the spinal fluid of patients suffering from GBS. Tr. at 162; Cornblath at 370. But the Cornblath study does not conclude that MBP is the target antigen in GBS – as Dr. Steinman admitted. *Id.* at 371; *see also* Tr. at 30-31, 162-63, 288-89. MBP is simply one of many other proteins and structures contained in peripheral myelin, and has been found to be released after the destruction of that myelin. Tr. at 180; Steinman, L. & Oldstone, M.B.A., *More Mayhem from Molecular Mimics*, 3 Nature Medicine 1322 (1997) (Pet'r's Ex. 20). Otherwise, none of the medical literature cited by Petitioner identifies MBP as a potential target of an immune response that triggers GBS. *See* Amato at 217 ("[t]he nature of the epitope is not known but probably is a glycolipid"); Komagamine at 394-95 (listing candidate target molecules but omitting MBP). Nor has Petitioner offered any instances in which MBP as the target antigen has been tested (at least in the context of a peripheral neuropathy like GBS).

---

substitutions (such as Y for F) would not necessarily hinder the activation of cross-reactive T cells with homologous protein sequences comprising MBP. Wucherpfennig at 1116, 1119-21.

Petitioner otherwise relied on studies establishing attacks on MBP pertaining to MS rather than peripheral neuropathies like GBS. Tr. at 283-86.[67]

Overall (and despite the fact that precise homology has not been required in other cases to successfully establish a medical theory in satisfaction of the first *Althen* prong), I found the evidence offered by Petitioner in support of her molecular mimicry theory to be less persuasive than Respondent's arguments to the contrary. However, even if I assume for sake of argument that Petitioner has offered just enough probative and reliable evidence to establish this first element of the causation test, such a determination would not aid her case. *Hirmiz*, 2014 WL 4638375, at *14. Because Ms. Blackburn has not demonstrated that she suffered from a vaccine-caused injury (give the above-discussed CIDP diagnosis and evidence that it predated her vaccination), the fact that the HPV vaccine could *in theory* cause a GBS variant other than the disease she actively suffered from is irrelevant to meeting her burden of proof.[68]

2. Prong Two: Did the HPV Vaccine Cause Ms. Blackburn's GBS and/or CIDP? – There is support in the treatment record establishing a "logical sequence of cause and effect" between the receipt of the HPV vaccine and the demyelinating symptoms Ms. Blackburn later suffered – assuming GBS were her illness, and that it arose only after the vaccination. At a minimum, the record contains statements by Ms. Blackburn's treating physicians that Petitioner's initially-diagnosed AIDP was caused by her vaccination – and although many of those opinions seem based on the timing between vaccine and injury, that fact alone does not render their opinions without value.

My determination that Ms. Blackburn's illness was more likely than not CIDP rather than GBS, and that it predated her vaccination, however, precludes a finding that the Gardasil vaccine likely caused her injury. This determination is based on expert testimony and the treating records, which clearly reflect that Petitioner's diagnosis was ultimately changed and has never been changed back since the time Ms. Blackburn received prednisone. The same evidence supports the conclusion that her illness more likely than not predated her vaccination, given the history of the neuropathic symptoms she has suffered.

Petitioner was unable to rebut the above. Thus, while she pointed out facts from the

[67] Those studies were also distinguishable for other reasons. For example, as Dr. Whitton noted, some involved experimentally-induced immune reactions (Tr. at 281-82) that were intended to produce central nervous system diseases like MS by significantly amplifying the intensity of the immune response – and therefore it was telling that they did not also produce a similar response in the peripheral nerves. *Id.* at 286-87, 325. Dr. Steinman himself ultimately admitted that EAE is not an acceptable model for GBS. *Id*. at 27, 169-70. At best, Dr. Steinman mentioned during his testimony an eighty year-old purported study, in which he claimed EAE-mice were found to have diseases of both the peripheral and central nervous system. *Id.* at 58. But that study was not filed in this case and has not otherwise been identified by Petitioner.

[68] I also note that Petitioner's *Althen* one theory does not address the idea of CIDP as being caused by the HPV vaccine – and indeed, Dr. Steinman strenuously challenged the accuracy of the CIDP diagnosis generally (even while asserting that his theory worked in either case).

treatment record that reasonably led Ms. Blackburn's treating providers to assume at the outset that she suffered from AIDP, she failed to explain the overall thrust of the record – and in particular the waxing and waning course of the disease she experienced – beyond some general references to "fluctuating" GBS. Nor did she rebut the effectiveness of the prednisone treatments, or the changed diagnosis. Dr. Steinman for his part was too selective in his review of the record, overemphasizing aspects of it that favor Petitioner's case while not effectively addressing the fundamental facts that point to CIDP as her real illness.

My analysis would be the same even if Petitioner had accepted the revised CIDP diagnosis but still argued that the HPV vaccination caused it. There is no question that Ms. Blackburn experienced a flare-up of her symptoms temporally after receiving the Gardasil vaccine – but it is well understood in Program cases that a mere temporal association between vaccination and illness does not establish causation. *Moberly*, 592 F.3d at 1323. As CIDP is clinically understood to involve a longer course, the relatively acute symptoms Petitioner experienced in the weeks after the vaccination are not consistent with the chronic nature of the illness, which would be slower to develop. And the neuropathic symptoms she experienced well before (and even the day of) her July 23rd vaccination also suggest that her disease preceded her vaccination.

3. Is there a Medically-Acceptable Temporal Relationship? – Less than a month passed between Ms. Blackburn's vaccination and the severe symptoms initially thought to be caused by AIDP. *See, e.g.*, Pet'r's Ex. 8 at 4-5. In addition, the record indicates that a few of her health care providers between August and October of 2009 believed (based on the information they possessed at the time) that temporally the vaccine likely had caused the symptoms they both observed and treated. *See*, *e.g.*, Pet'r's Ex. 4 at 9. Certainly there is support (in both the relevant medical literature as well as the decisions of other special masters) for the conclusion that the timeframe between vaccination and Ms. Blackburn's temporally-subsequent symptoms was medically acceptable – again, assuming she suffered from GBS. *See, e.g.*, *Corder v. Sec'y of Health & Human Servs.,* No. 08-228V, 2011 WL 2469736, at *27-29 (Fed. Cl. Spec. Mstr. May 31, 2011) (proposed four-month onset period from vaccination to GBS too long; two months is longest reasonable timeframe).

But Petitioner has not established that she more likely than not suffered from GBS. And even if Petitioner had accepted the CIDP diagnosis and argued that it was caused by the July 2009 vaccination (rather than predating it), there would still be a lack of a medically-acceptable temporal relationship, due to the fact that (as none of the parties disputed) the course of CIDP is considerably longer than AIDP. CIDP would less likely become as acute (as the records establish Ms. Blackburn's illness was) within a month of vaccination, and no evidence offered by Petitioner or her expert alters this conclusion. Thus, Petitioner's claim also founders on her inability to meet the third *Althen* prong.

CONCLUSION

I have great sympathy for the medical problems and related suffering Ms. Blackburn has experienced over the past several years. However, a petitioner who fails to demonstrate by a preponderance of the evidence that the vaccine she received was more likely than not the cause of her illness is not entitled to compensation. Such is the case here. Because Ms. Blackburn has failed to demonstrate that she in fact suffered from injuries caused by her receipt of the HPV vaccine, I DENY an entitlement award in this case. I instruct the Clerk of Court to enter judgment dismissing the case unless a motion for review is filed.

IT IS SO ORDERED.

s/Brian H. Corcoran
Brian H. Corcoran
Special Master